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# Annals of Otology, Rhinology and Laryngology

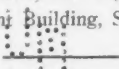
FOUNDED BY JAMES PLEASANT PARKER

INCORPORATING  
THE INDEX OF OTOLARYNGOLOGY

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L. W. DEAN, M. D., Editor-in-Chief  
Kingshighway and Euclid Ave., St. Louis

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I.

SOME EFFECTS IN LATER LIFE OF OTITIS MEDIA  
IN INFANCY.\*

By C. C. BUNCH, PH. D., AND R. C. GROVE, M. D.,

BALTIMORE.

The purpose of this study is to determine the effects of otitis media occurring in infancy on the acuity of hearing in later life. Material for similar studies is available in all pediatric clinics, since acute suppurative otitis media is an extremely frequent complication in the hospitalization of infants. It seems to us that valuable information in the problem of preventing deafness can be obtained by reexamining from year to year after their discharge from the hospital as many of these children as possible and carrying out whatever dietetic and operative measures may be necessary to stop any chronic aural suppuration that may persist in a few and, when they are seven or eight years of age, to test them routinely with voice, tuning forks and audiometers to determine the degree and exact location in the tonal range of any defect in hearing that may be present.

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\*From the Otological Research Laboratory, Johns Hopkins University.

Such a study differs materially from those conducted by Newhart, Fowler, Shambaugh and others. Their investigations were conducted to segregate those children who had defective hearing in order that proper adjustment in the educational scheme might be made and therapeutic measures recommended. Our plan, on the other hand, is to secure accurate information regarding the impairment of hearing, if any, that may be present from seven to fifteen years after the evidence of a definite suppurative otitis media which was observed and treated in the hospital, and by X-rays of the mastoids to determine the effect of this middle ear infection on the pneumatization of the mastoid.

This first report is made on a group of children who were not followed from year to year, but were selected by reviewing the hospital histories as determined below, traced by the usual social service methods,\* and have so far had only one examination of their auditory acuity.

Since the inauguration of the department of pediatrics in the Johns Hopkins Hospital in 1912, complete clinical histories of all cases admitted have been preserved. With the consent and cooperation of this department some twenty thousand case histories were reviewed and those selected which were considered desirable, and the children, eighty in number, were brought to the hospital for examination.

The nature of the information desired necessitated certain requisites in the selection of cases. First, children under seven or eight years of age do not usually have sufficient mental development to respond accurately to rather long and complicated hearing tests, especially if the tests are conducted under new and strange environment. Therefore, in reviewing the hospital histories, only children who had an admission date prior to 1921 were selected. This gave a minimal age of seven years at the time of this examination. Second, an attempt was made to secure only those who had had a severe otitis media

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\*We wish to express our appreciation of the interest and assistance of the Misses Ann Follis, McQueen Gibbs and Carolyn Griswold in reviewing the hospital histories, selecting the desired cases and securing the return of the children for examination.

We also wish to acknowledge the aid of Dr. S. J. Crowe, who made it possible for this study to be inaugurated and carried to completion and who has at all times aided and encouraged us in the work.

or recurring attacks. All clinical evidence of acute otitis media in infants usually disappears after a myringotomy, although repeated myringotomies or drainage of the mastoid may be necessary. In order to be sure that the cases selected should be those with maximal trauma, only those with a history of having had three or more myringotomies were chosen. This arbitrary standard may not represent the extent of the middle ear involvement, but it at least gave an indication of the degree of trauma to the drum and middle ear. The ages of the children selected ranged at the time of examination in 1928-29 from seven to sixteen years. Their ages at the time of admission to the hospital ranged from two days to four years and eight months. Nine were over two years and forty-seven were under one year of age.

The examination consisted of accurate hearing tests made with the 1A and 4A Western Electric audiometers, tuning forks and the monochord; second, of careful otoscopic examination by members of the clinical staff of the department of otolaryngology; and third, since the development of the mastoids could so easily be studied in the group, X-rays were taken in fifty-two cases. In addition to this, the children or their parents were carefully questioned to secure information concerning the condition of the ears and the hearing since the time of hospitalization.

The results of the tests with the 1A audiometer are arranged in the form of a scattergram and shown in Fig. 1. The numbers in the horizontal column indicate the pitch of the thirteen tones used as stimuli in the tests and those in the vertical column correspond to the intensity readings of the audiometer. Each dot in the scattergram represents a threshold for one ear. It will be seen that the thresholds show a considerable intensity range, but this was to be expected, since thirty ears gave records which indicated definite loss of acuity.

Fig. 2 shows the means and mean variations calculated on the basis of these threshold observations, the solid line being the means and the lines of dashes the mean variations.

The records of the thirty ears which were considered hard of hearing were eliminated from the group, and the scattergram for the records of the better hearing ears is shown in Fig. 3. The wide scattering for the tones under 8192 d. v. is

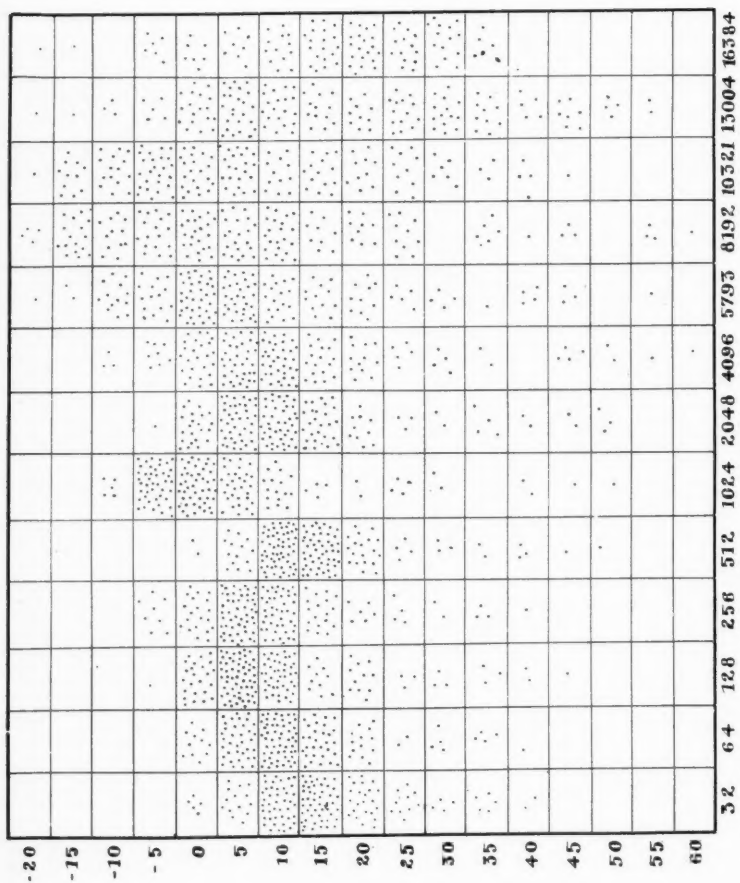


Fig. 1. Scattergram showing threshold observations for all children in the group. Each dot is the threshold for one ear. The numbers in the vertical column are the intensity readings taken from the audiograms; those in the horizontal column indicate the pitch of the tones tested.

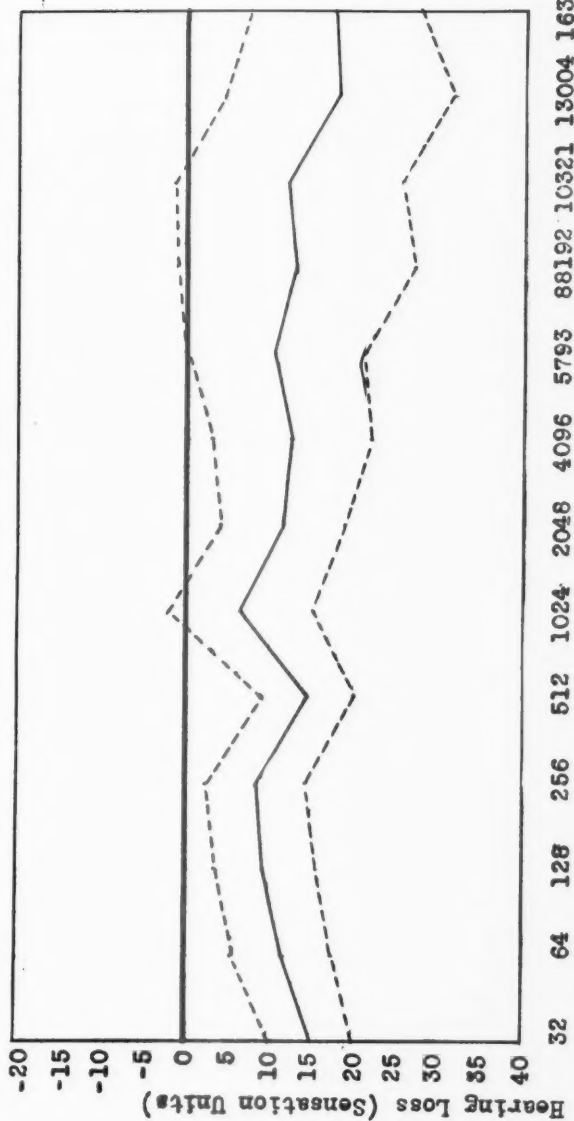


Fig. 2. The heavy broken line shows the means and the lines of dashes the mean variations calculated from the thresholds given in Fig. 1.

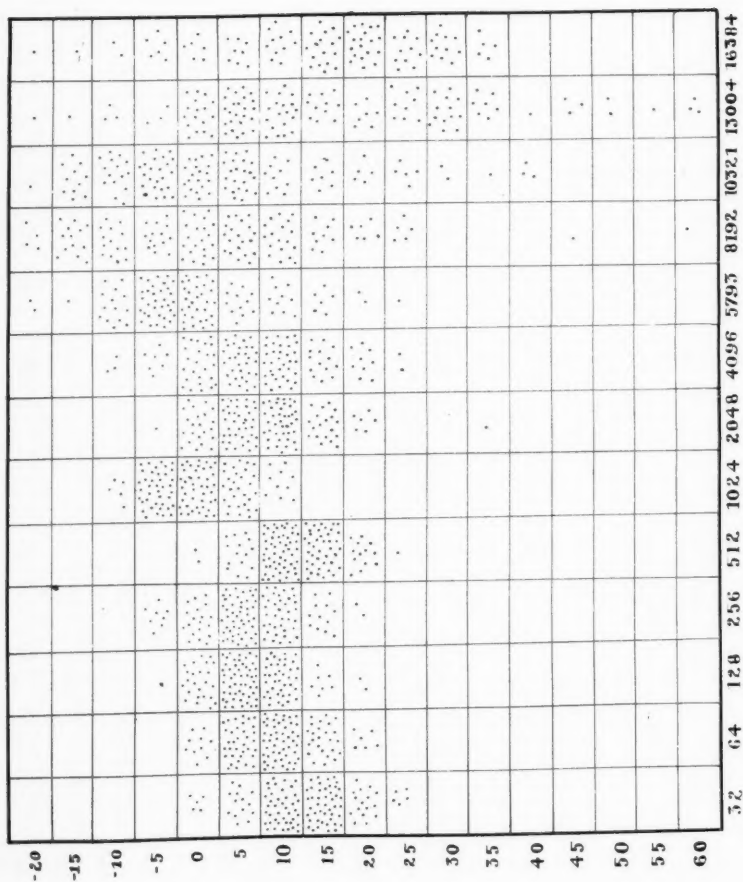


Fig. 3. Scattergram of thresholds for the 130 better hearing ears.



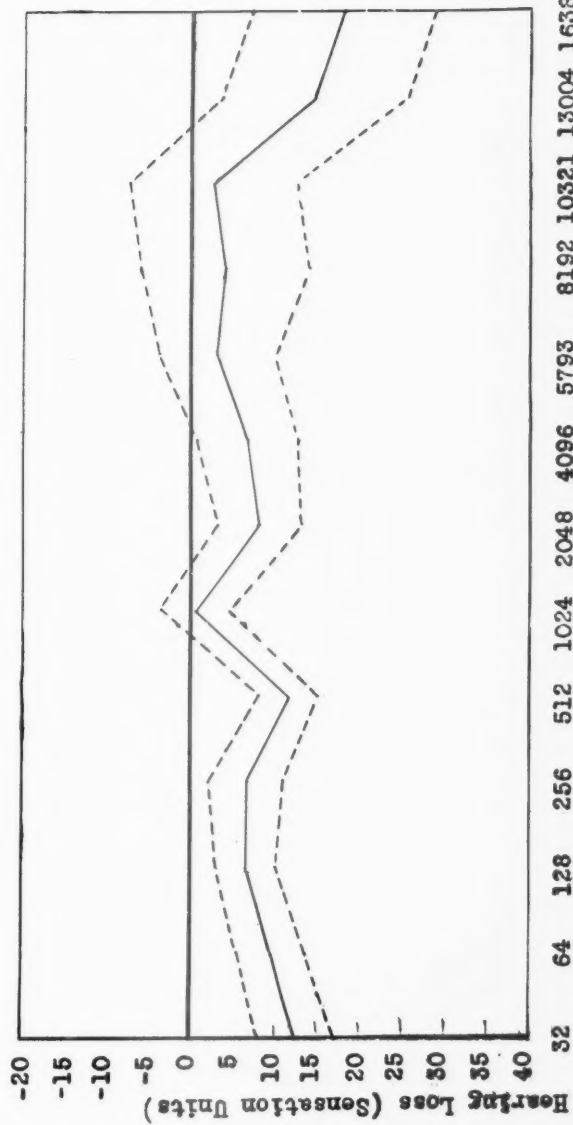


Fig. 4. Means and mean variations calculated from Fig. 3.

practically eliminated. The thresholds for each tone would fall statistically into quite a normal curve of distribution. At 8192 d. v. and for tones of higher pitch, the scattering is about equally marked in both Figs. 1 and 3.

The means and mean variations, calculated on the basis of the thresholds of the better hearing ears, are shown in Fig. 4. Here again the solid line represents the means and the lines of dashes the mean variations.

One of us in an earlier study\* showed that the calculated averages of records secured from tests of a large number of hospital patients who have no subjective or objective evidence of deafness give distinct differences in successive decades. It was not assumed in this earlier study that these patients had absolutely "normal" hearing, but it was thought that they were representative. It cannot be assumed in the present study that these better hearing children had "normal" hearing, for all had had otitis media with considerable trauma to the tympanic membrane several years prior to the time these tests were made. But for purposes of comparison, the averages secured from the twenty-year group of this earlier study is shown in Fig. 5, together with these two groups, all plotted to the same scale.

It will be noted that the averages of the thresholds for the better hearing children are higher than the averages for the thresholds of the twenty-year group—i. e., they hear the tones at fainter intensities. This is true for all tones except 13004 d. v. and 16384 d. v. The highest tone, 16384 d. v., was inaudible at its maximum intensity to twenty-nine ears in this group. If the records of all the children be considered, it will be noted that the threshold is better for the children than for the twenty-year group for tones under 4096 d. v. Above this tone the average of the twenty-year group is better than that for the children. This seems contrary to the teaching that middle ear lesions are characterized by a loss of acuity for low tones, for here the average for the children who had otitis media shows fainter thresholds for low tones than the average of a twenty-year group with supposedly "normal" hearing.

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\*Bunch, C. C.: Age Variations in Auditory Acuity. Arch. of Otolaryng., Vol. 9, pp. 625-636.

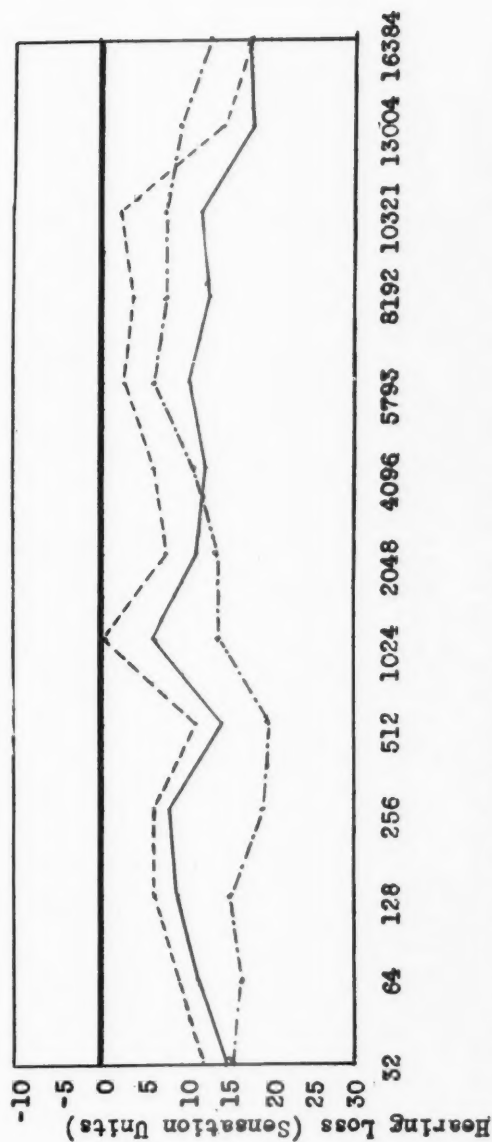


Fig. 5. The means from Figs. 2 and 4 compared with the means secured from a group between 20 and 30 years of age. The solid line is taken from Fig. 2, the line of dashes from Fig. 4, and the line of dashes and dots from a group between 20 and 30 years of age.

For tones above 4096 d. v. the average for the twenty-year group is distinctly better.

No mean has been calculated for those in the group who were definitely hard of hearing, since they are but thirty in number, a group too small to be considered statistically. A comparison of the scattergrams in Figs. 1 and 3 shows how the records from the ears which were hard of hearing affects the scattering of the thresholds.

The tests with the 4A (phonographic) audimeter showed that twenty-one ears had a hearing loss of 6 units or more, twenty-four had a loss of 3 units, which is by most investigators considered to be the equivalent of a fluctuation in attention. The greatest loss encountered was 30 units in an ear with a large perforation in the tympanic membrane and discharge at the time these tests were made.

Tests for determining the upper limit with the monochord were made on one hundred and twenty-eight ears. The results are illustrated graphically in Fig. 6. It will be seen that the peak of the curve for air conduction is at 17000 d. v., while that for bone conduction is at 18000 d. v. Forty-seven ears had the upper limit for bone conduction 2000 d. v. or more higher than the upper limit for air conduction, forty-eight had bone conduction 1000 d. v. higher in pitch and twenty-three had the same upper limit for both bone and air conduction. Eight ears with perforations still present, which were tested with the monochord, all had the upper limit for bone conduction 1000 d. v. or more higher than that for air conduction.

A group of one hundred sixty-four young adults with supposedly "normal" hearing which were previously tested by one of us\* showed somewhat different findings. Approximately 70 per cent in this earlier group had the same threshold for both bone conduction and air conduction, the peak of the curves for these observers for both bone conduction and air conduction being at 19000 d. v. This adds evidence to the assumption that conductive lesions tend to lower the upper limit of audibility, especially for sounds transmitted by air.

\*Bunch, C. C.: Functional Hearing Tests in Normal Cases. *ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY*, 1924, pp. 1-19.

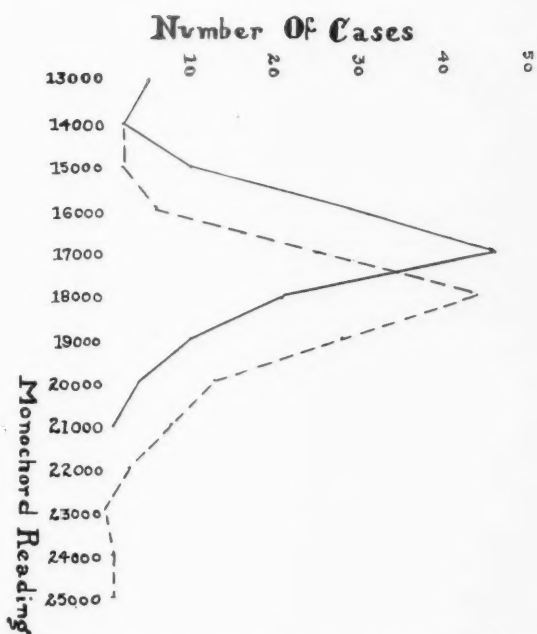


Fig. 6. Upper limit observations for air conduction and bone conduction as determined by the monochord.

Eight cases had discharging ears at the time of examination, two were bilateral and six unilateral. The Rinne test—i. e., the comparison of the relative bone conduction and air conduction, done with the weighted  $c^2$  fork of the Bezold series, gave a negative response in both ears in these bilateral cases, and the Weber test made with the same fork gave a lateralization to the ear with the poorer hearing in one case, and in the other the sound was not lateralized to either side. The six cases with unilateral discharge gave a negative response to the Rinne test with the affected ears in all but one, and the Weber test was lateralized to the affected ear in all but one. The five cases with dry perforations, all of which were unilateral, gave a positive response to the Rinne test in all but one, and the Weber test gave a lateralization to the affected side in two cases and was not lateralized in three.

The Rinne and Weber tests were done on sixty-one other cases. The negative response to the Rinne test was given but three times. The Weber test was lateralized to one side in eleven cases, in three toward the side with the better hearing, in three toward the side with the poorer hearing, and five lateralized the sound when the hearing was equal in both ears.

The appearance of the tympanic membrane and middle ear determined by the otoscopic examination gave little hint as to the hearing power except in cases of frank discharge. Dry perforations were not always accompanied by appreciable loss of acuity of hearing.

If in individual tests with the phonographic audiometer it is possible for children with dry perforation to secure records showing practically normal hearing, it seems probable that in group tests where a loss of six units or more call for additional examination, such cases as the first two mentioned above may easily be overlooked.

Twelve tympanic membranes were listed as normal. Of these, with the phonographic audiometer, ten showed no loss for voice, one showed a loss of 3 units and one of 6 units. The description of the others varied from "slight retraction," "thinned" and "slight scarring" to "very opaque," "retracted, scarred and thrown in folds" and "calcified plaques." One listed as "slightly retracted" had a loss of 15 units. One with a "calcified plaque" had a loss of 6 units, while another with

the same description had no loss. Another listed as having "an unusual amount of scarring" had 9 units loss, and one listed as having "moderate retraction" had an equal loss. One listed as "quite retracted and opaque" had no loss, while another listed as "very opaque" had a loss of 6 units. Of the ears with normal tympanic membranes, one had had five myringotomies while in the hospital, two had had three, five two, three one, and one was a unilateral case where one ear had not been affected. One case with seven myringotomies on one side and eight on the other had 3 units loss on one side and 9 on the other, and both tympanic membranes were listed as having "moderate retraction." The otoscopic examinations were made by several clinical men; however, one of us (R. C. G.) examined forty-eight cases. The reports from the different examiners were quite consistent.

The study of the development of the mastoids by means of X-rays was not begun until after twenty-three cases had been examined otherwise. A few refused. The cases were not selected. The X-ray examination was routine. Roentgenograms were secured in fifty-two cases. These were interpreted and reported to the laboratory by Dr. F. H. Baetjer. In these reports, fifty-two are listed as sclerosed, four as being slightly sclerosed, one with clouding, four not completely developed, four involved, and thirty-one normal.

To arrive at statistical conclusions from the X-ray study is almost as difficult as from the otoscopic examination. For example, one child had both ears involved when eight months of age, the infection lasting over a period of six months. Six myringotomies had been done on the right and three on the left. According to the X-ray reports, the mastoid development on the right was normal and there was slight sclerosis on the left. Another child had both ears involved at two years of age, the infection lasting for two months. Eight myringotomies were done on the right and seven on the left, and the X-rays showed normal development of the mastoids. Another case entered the hospital with spontaneous rupture of the right drum membrane while the left was reddened. The infection cleared up in five days. No myringotomies were done but the X-rays showed sclerosis of both mastoids. Another child was four and a half months old at the time of entrance to the hos-

pital, and the history stated that there had been a recurrence of ear infection over a period of three years. The right ear was discharging at the time of admission. The left ear became involved and four myringotomies were done on it. The X-rays show the left ear to be clear and the right not developed. Another child was five and a half months of age and the infection extended over a period of four months. Five myringotomies were done on the right side and four on the left, and the mastoids show normal development. The youngest child in the series was two days old when admitted to the hospital in 1919. Both ears were involved and the infection lasted for one month. Five myringotomies were done on the right and four on the left, and the X-rays, taken in April, 1929, show that the right mastoid is clear and the left shows partial clouding. Another child, one year of age, had bilateral otitis for one week with two myringotomies on the right and one on the left, but both mastoids now show sclerosis.

Of the cases which were discharging at the time of this examination who had X-ray of the mastoids taken, three in number, two were listed as sclerosed and one was not completely developed. X-rays of the other five were not made. Of those with dry perforations, one was listed as showing old infection in the X-ray picture, one was sclerosed, one slightly sclerosed and one clear.

For more complete illustration, the audiograms, X-ray pictures, otoscopic notes and details of the histories of twenty selected cases are given below.

#### SUMMARY.

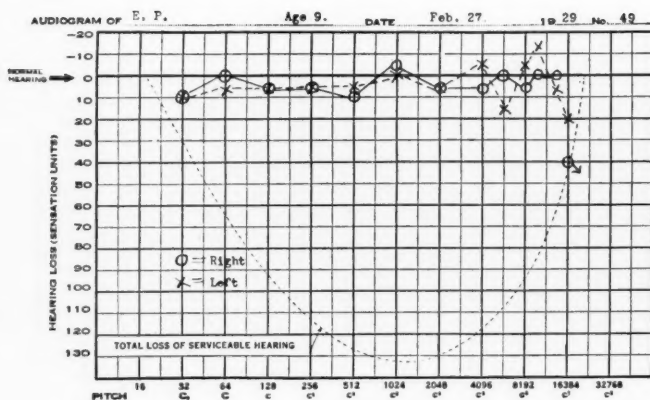
A group of children ranging in age from seven to sixteen years who, according to their hospital histories, had otitis media in infancy, were returned to the Johns Hopkins Hospital for otologic examination during the period from October, 1928, to June, 1929. X-ray plates showing the development of the mastoids were made in fifty-two cases. The cases were selected for examination on the basis of the fact that during their hospitalization they had had repeated myringotomies. Thirty ears, or 19 per cent, had appreciable hearing loss when examined. Ten of these thirty were discharging. Dry perforations were present in five. The tympanic mem-



branes do not necessarily, after a period of years, present evidence of repeated myringotomies, since in twelve ears of this group the tympanic membranes were normal. The otoscopic examination, except in cases where discharge was present, gave the examiner little idea as to the relative hearing power. Cases are presented which show that factors other than otorrhea alone must play an important part in the pneumatization of the mastoid and in the loss of acuity where deafness exists. A severe otitis media in infancy does not necessarily result in the arrest of the process of pneumatization of the mastoid. The X-rays of the mastoids can be interpreted only in conjunction with clinical evidence, as an extensive pneumatization may be present after an otitis media of five years' duration, while, on the other hand, dense sclerosis may be present after one of but four months' duration. The X-rays showed normal development in thirty-one mastoids. Sixty-seven were reported "sclerosed" or with some other form of arrested pneumatization.

## REMARKS.

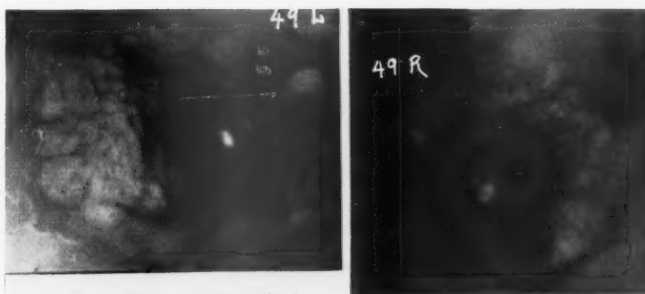
The health records of the children before admission to the hospital and for the interval between their hospitalization and the present time are unknown factors in this study. The statements of both parents and children were repeatedly shown to be inaccurate. The various systemic conditions which often accompany middle ear infections have not been investigated, since this study is restricted in its scope chiefly to the problems of hearing.



E. P. Age 9. Feb. 27, 1929

HEARING	RIGHT EAR				LEFT EAR				HEARING
Loss	1	2	3	4	5	6	7	8	Loss
30	54	56			48	55			30
27	41	68			23	54			27
24	84	63			66	88			24
21	51	14			41	42			21
18	18	34			81	23			18
15	23	12			68	85			15
12	22	31			21	86			12
9	11	63			15	64			9
6	48	88			43	58			6
3	34	-1			31	3-			3
0	-6	-6			-4	1-			0
-3	-1	12			5-	-			-3
HEARING LOSS.....					HEARING LOSS.....				

CASE 49.



Otitis media, bilateral, for at least 18 months.

Age at onset, 12 months.

Tympanic membrane on right was incised 4 times and on the left 5 times.

No aural infection during the past 6 years.

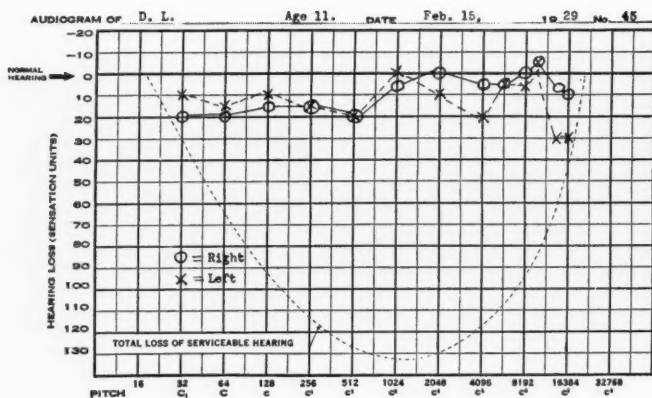
Tympanic membranes at present are intact, not retracted, have good light cones but show healed perforations on each side.

The 1A and 4A audiometers show approximately normal hearing.

AC is greater than BC on each side for c2 fork.

X-rays: Extensive pneumatization of both mastoids.

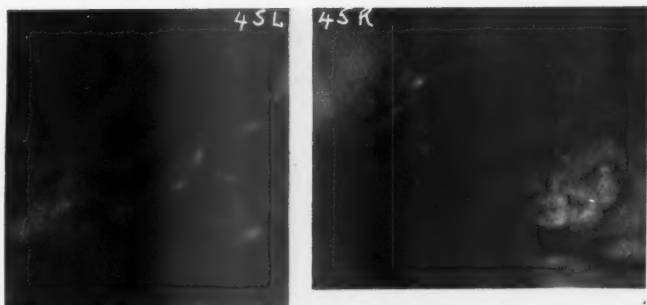
CASE 49.



D. L. Age 11. Feb. 15, 1929

HEARING LOSS	RIGHT EAR				LEFT EAR				HEARING LOSS
	1	2	3	4	5	6	7	8	
30	54	56	85		48	55			30
27	41	68	83		23	54			27
24	84	63	42		66	88			24
21	51	14	18		41	42			21
18	18	34	61		81	23			18
15	23	12	35		68	85			15
12	22	31	32		21	36			12
9	11	23	41		15	84			9
6	48	88	51		43	58			6
3	31	—	—		11	23			3
0	11	—	—		24	—			0
-3	—	—	—		56	11			-3
HEARING LOSS _____					HEARING LOSS _____				

CASE 45.



Otitis media, bilateral, for 4 years, more severe on the left.

Age at onset, 10 months.

Tympanic membrane on the right was not incised; left incised 3 times.

No aural infection during the past 6 years.

The tympanic membranes at present are intact, not retracted and have good light cones. There is a healed perforation in the left.

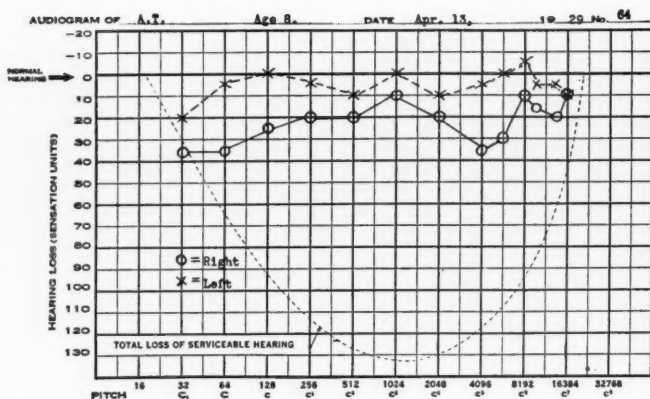
The 1A audiometer shows a possible slight loss for high tones on the left.

The phonographic audiometer shows 3 units loss for voice on the right and no loss on the left.

AC is greater than BC on each side for the c2 fork.

X-rays: Extensive pneumatization of both mastoids.

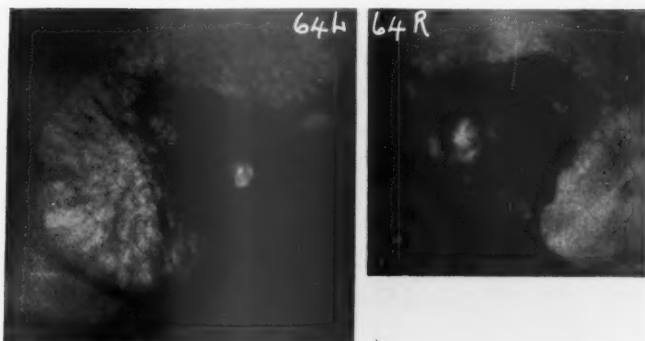
CASE 45.



A.T. Age 8. Apr. 13, 1929

HEARING Loss	RIGHT EAR				LEFT EAR				HEARING Loss
	1	2	3	4	5	6	7	8	
30	54	56			48	55			30
27	41	68			23	54			27
24	84	63			66	88			24
21	57	14			41	42			21
18	14	34			81	23			18
15	23	12			68	85			15
12	2	31			21	86			12
9	11	63			15	64			9
6	4	—			48	58			6
3	—	—1			41	38			3
0	—	—			—4	18			0
-3	54	—			56	—			-3
HEARING LOSS _____					HEARING LOSS _____				

CASE 64.



Otitis media, bilateral, for a least 3 years on the left and intermittently for 8 years on the right.

Age at onset,  $4\frac{1}{2}$  months.

Tympanic membrane on the right incised 4 times and on the left 3 times.

No aural infection on the left during the past  $4\frac{1}{2}$  years but right has discharged intermittently to present time.

Tympanic membrane on the left at present is intact, not retracted and has a good light cone. The right shows a perforation in the anterior inferior segment with granulations in the middle ear.

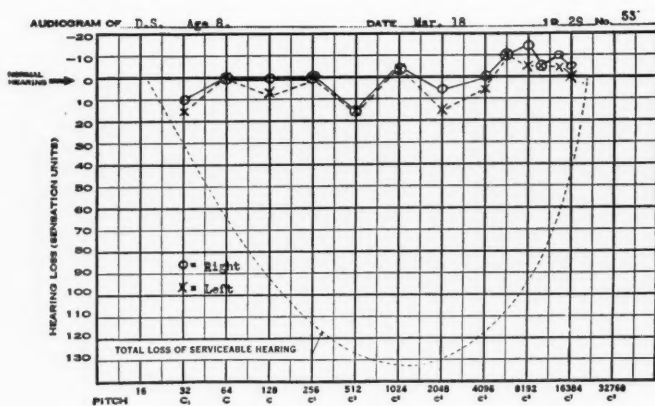
The 1A audiometer shows approximately normal hearing on the left and a slight impairment for all tones on the right.

The phonographic audiometer shows normal hearing for voice on the left and 6 units loss on the right.

AC is greater than BC on the left. BC is greater than AC on the right for c2 fork.

X-rays: Extensive pneumatization of the left mastoid and clouding of the right.

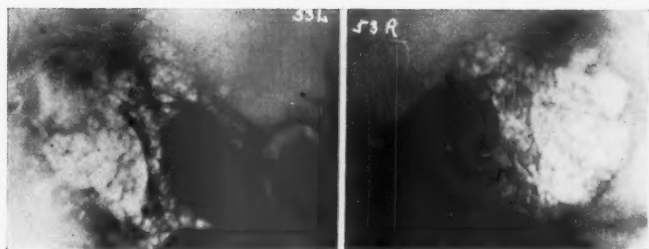
#### CASE 64.



D.S.		Age 8.				Mar. 18, 1929			
HEARING	RIGHT EAR				LEFT EAR				HEARING
LOSS	1	2	3	4	5	6	7	8	LOSS
30	54	56			48	55			30
27	41	68			23	54			27
24	84	63			66	88			24
21	51	14			41	42			21
18	18	34			11	23			18
15	23	12			68	85			15
12	22	31			21	86			12
9	11	63			15	64			9
6	48	88			43	58			6
3	31	81			31	38			3
0	XX	26			—	18			0
-3	5X	1X			6X	23			-3
HEARING LOSS _____					HEARING LOSS _____				

CASE 53.





Otitis media, bilateral, for at least 21 months.

Age at onset, 1 year.

Tympanic membrane on the right incised 2 times and on the left 3 times.

No aural infection during the past 5 years.

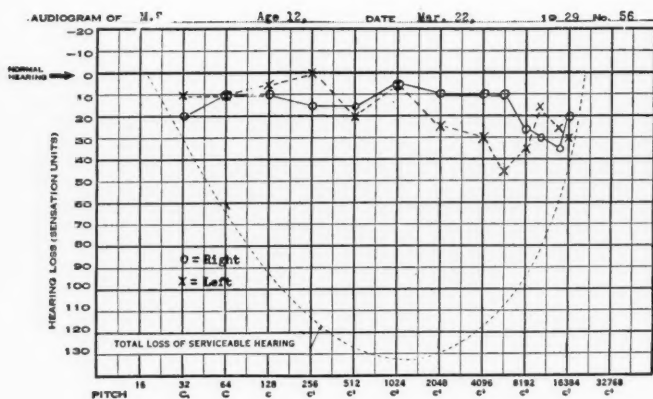
Tympanic membranes at present are intact, not retracted and have good light cones

The 1A and 4A audiometers show approximately normal hearing.

AC is greater than BC on both sides for the c2 fork.

X-rays: Extensive pneumatization of both mastoids.

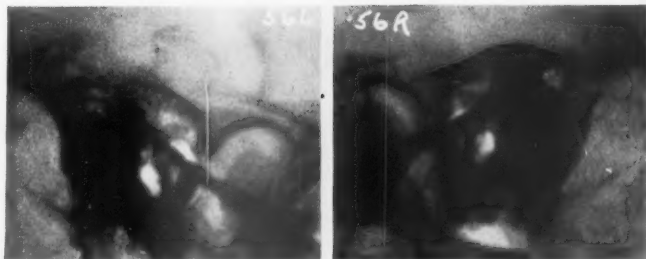
CASE 53.



M.S. Age 12. Mar. 22, 1929

HEARING Loss	RIGHT EAR				LEFT EAR				HEARING Loss
	1	2	3	4	5	6	7	8	
30	54	56			48	55			30
27	41	68			23	54			27
24	24	63			66	88			24
21	51	14			41	42			21
18	18	34			81	23			18
15	23	12			68	85			15
12	22	31			21	86			12
9	11	63			15	64			9
6	48	XX			4X	58			6
3					—	—			3
0					—	1-			0
-3					5-				-3

HEARING LOSS. HEARING LOSS.



Otitis media, bilateral, for at least 2 years, more severe on the left.

Age at onset,  $3\frac{1}{3}$  years.

Tympanic membrane on the right was incised once and on the left twice.

No aural infection during the past  $6\frac{1}{2}$  years.

Tympanic membranes at present show a dry perforation on the left and a calcified plaque on the right.

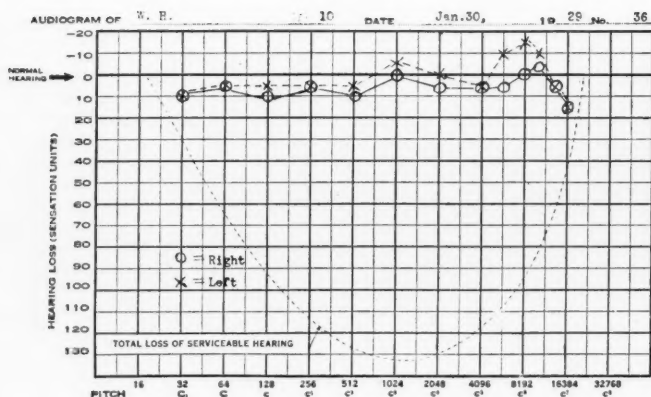
The 1A audiometer shows slight impairment for high tones on both sides, more marked on the left.

The phonographic audiometer shows approximately 3 units loss for voice on each side.

AC is greater than BC on both sides for the c2 fork.

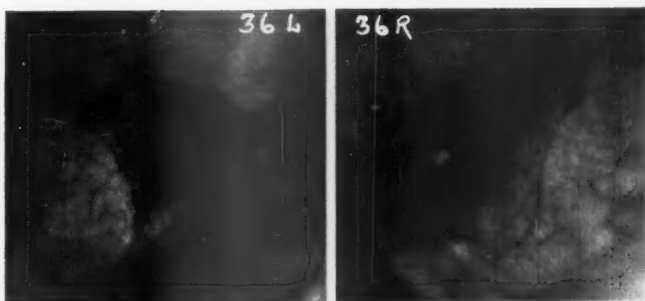
X-rays: Clouding of both mastoids.

CASE 56.



W. H.		Age 10				Jan. 30, 1929				
HEARING		RIGHT EAR				LEFT EAR				HEARING
Loss	1	2	3	4	5	6	7	8	Loss	
30	54	56			48	52			30	
27	41	68			23	54			27	
24	84	63			66	88			24	
21	51	14			41	42			21	
18	18	34			81	23			18	
15	23	12			68	85			15	
12	22	31			21	86			12	
9	11	63			15	64			9	
6	48	88			43	88			6	
3	34	81			31	34			3	
0	—	—			—	18			0	
-3	51	—			56	—			-3	
HEARING LOSS					HEARING LOSS					

CASE 36.



Otitis media, bilateral, for at least 9 months.

Age at onset, 8 months.

Tympanic membrane on the right was incised 6 times and on the left 5 times.

No aural infection during the past  $8\frac{1}{2}$  years.

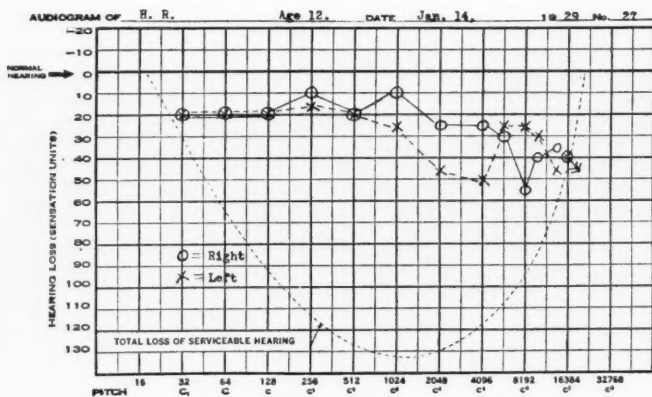
Tympanic membrane at present on the right is intact, not retracted and has a good light cone. The left one is retracted, atrophic and contains two small dry perforations.

The 1A and 4A audiometers show approximately normal hearing.

AC is greater than BC on both sides for the c2 fork.

X-rays: Extensive pneumatization of both mastoids.

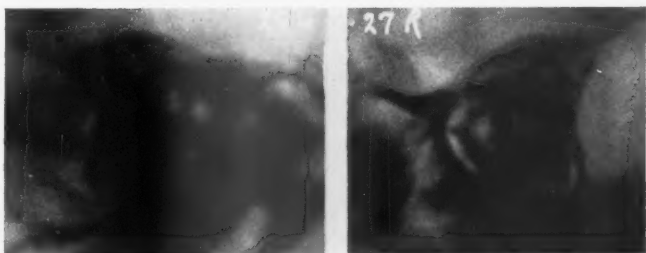
CASE 36.



H. R. Age 12. Jan. 14, 1929

HEARING	RIGHT EAR				LEFT EAR				HEARING
LOSS	1	2	3	4	5	6	7	8	LOSS
30	54	56	85		48	55			30
27	41	68	83		<del>48</del>	54			27
24	84	63	42		66	88			24
21	51	14	18		41	42			21
18	18	34	61		81	23			18
15	23	12	35		68	85			15
12	—	31	<del>84</del>		<del>48</del>	86			12
9	11	63	44		15				9
6	48	<del>88</del>	<del>54</del>						6
3	—	<del>84</del>							3
0	—								0
-3	—								-3
HEARING LOSS _____					HEARING LOSS _____				

CASE 27.



Otitis media, bilateral, for at least  $7\frac{1}{2}$  years.

Age at onset, 9 months.

Tympanic membrane on the right was incised 8 times and on the left 7 times.

No aural infection during the past  $4\frac{1}{2}$  years.

Tympanic membrane at present on the right is retracted and shows a dry perforation occupying both posterior segments. The left is intact, retracted, and shows two healed perforations anteriorly.

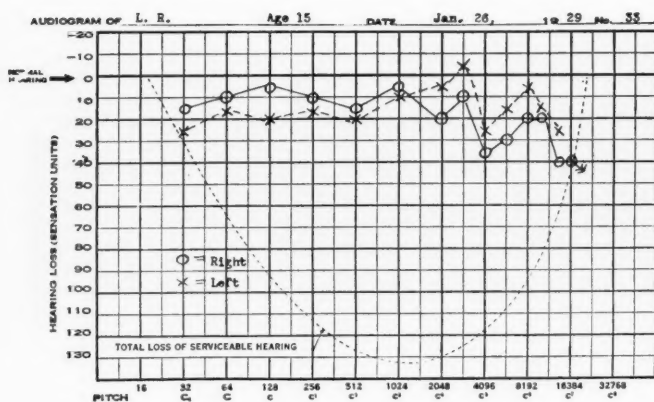
The 1A audiometer shows a loss for high tones on each side.

The phonographic audiometer shows 3 units loss on the right and 9 units loss for voice on the left.

AC is greater than BC on the right and BC is greater than AC on the left for the c2 fork.

X-rays: Clouding of both mastoids, more on the left.

CASE 27.

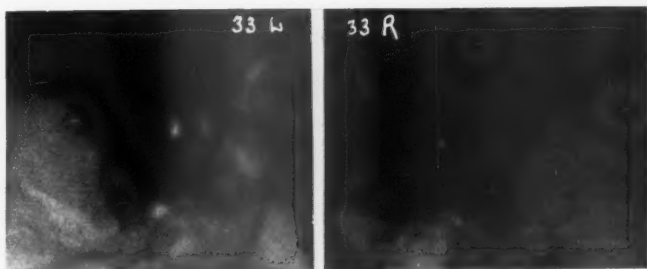


L. R. Age 15 Jan. 26, 1929

HEARING LOSS	RIGHT EAR				LEFT EAR				HEARING LOSS
	1	2	3	4	5	6	7	8	
30	54	56			48	55	48		30
27	41	68			23	54	63		27
24	84	63			66	88	83		24
21	51	14			41	42	32		21
18	18	24			81	23	85		18
15	33	12			68	85	63		15
12	—	31			21	86	18		12
9	11	63			15	64	24		9
6	48	—			48	58	—		6
3	—	—			—	38	25		3
0	—	—			—	—	—		0
-3	—	—			—	—	—		-3
HEARING LOSS					HEARING LOSS				

CASE 33.





Otitis media, bilateral, for at least 4 months.

Age at onset, 28 months.

Tympanic membrane on the right was incised twice and on the left three times.

No aural infection during the past 12 years.

Tympanic membranes at present are intact, not retracted and have good light cones.

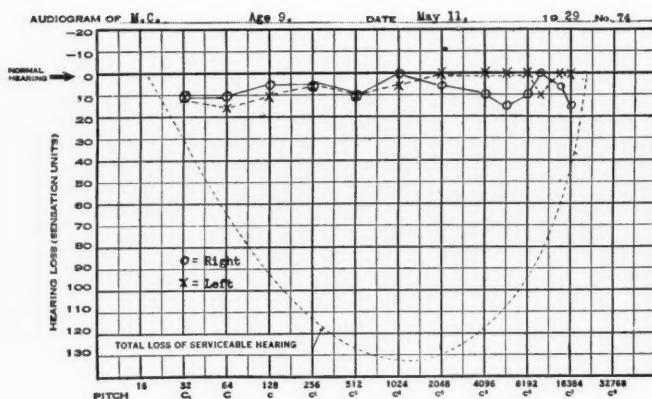
The 1A audiometer shows impairment for all tones, but more marked for the higher frequencies.

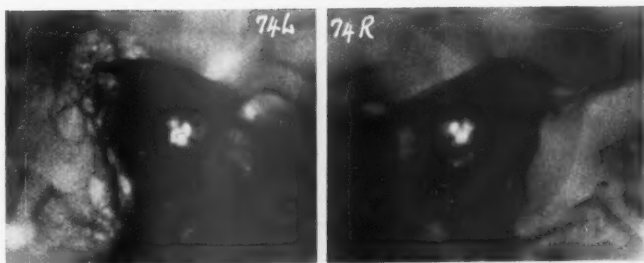
The phonographic audiometer shows 6 units loss for voice on the right and 3 units loss on the left.

AC is greater than BC on the right and BC is greater than AC on the left for the c2 fork.

X-rays: Dense sclerosis of both mastoids.

#### CASE 33.





Otitis media, bilateral, for at least 2 years.

Age at onset, 14 months.

Tympanic membrane on the right was incised 3 times and on the left 4 times.

No aural infection during the past 6 years.

Tympanic membranes at present are intact, not retracted and have normal light cones.

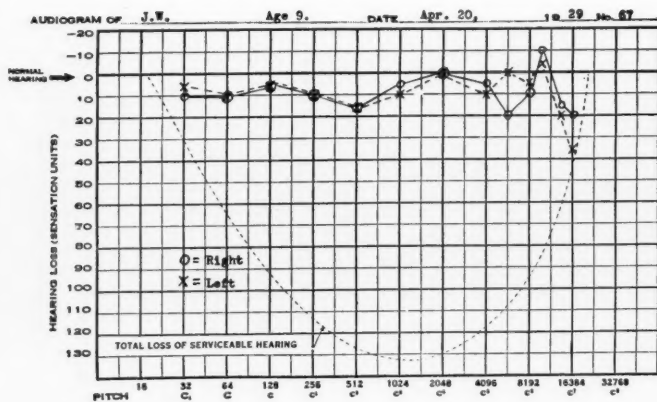
The 1A audiometer shows some impairment for high tones on the right.

The phonographic audiometer test is normal.

AC is greater than BC on both sides for the c2 fork.

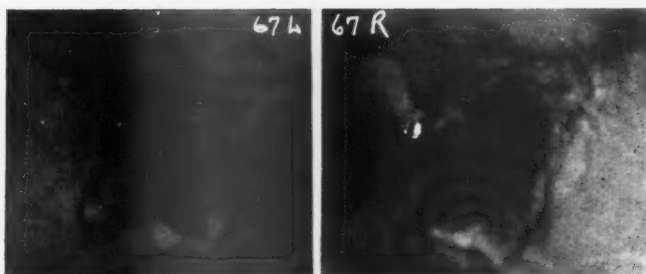
X-rays: Note the difference in pneumatization between the two sides. Is there any correlation between the loss of high tones and the impaired pneumatization on the right?

CASE 74.



J.W.		Age 9.				Apr. 20, 1929					
HEARING LOSS	RIGHT EAR				LEFT EAR				HEARING LOSS		
	1	2	3	4	5	6	7	8			
30	54	56			48	55			30		
27	41	68			23	54			27		
24	84	63			66	88			24		
21	51	14			41	42			21		
18	18	34			11	23			18		
15	23	12			68	85			15		
12	22	31			21	86			12		
9	11	63			15	64			9		
6	48	88			43	58			6		
3	38	81			31	58			3		
0	28	6			24	—			0		
-3	58	8			56	—			-3		
HEARING LOSS.....					HEARING LOSS.....						

CASE 67.



Otitis media, bilateral, for at least 5 months.

Age at onset, 5½ months.

Tympanic membrane on the right was incised 5 times and on the left 4 times.

No aural infection during the past 8 years.

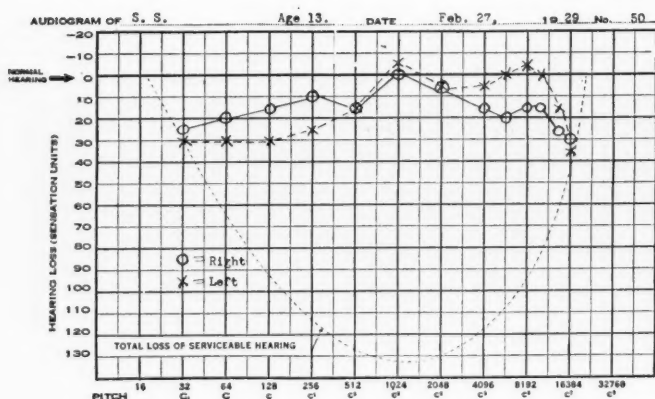
Tympanic membranes at present are intact, retracted and have good light cones.

The 1A and 4A audiometers show approximately normal hearing.

AC is greater than BC on both sides for the c2 fork.

X-rays: Extensive pneumatization of both mastoids.

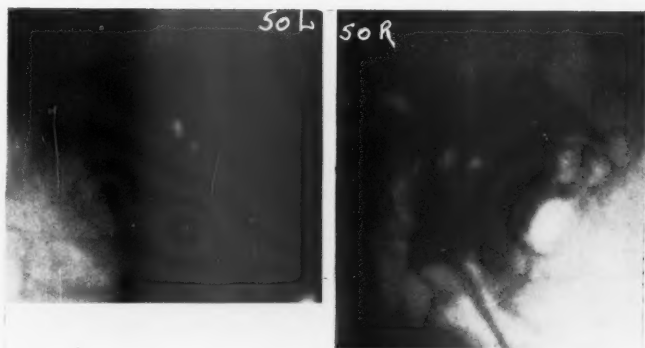
#### CASE 67.



S. S. Age 13. Feb. 27, 1929

HEARING LOSS	RIGHT EAR				LEFT EAR				HEARING LOSS
	1	2	3	4	5	6	7	8	
30	54	56			48	55			30
27	41	68			23	54			27
24	84	63			66	88			24
21	51	14			41	42			21
18	18	34			81	23			18
15	23	12			68	85			15
12	22	31			21	86			12
9	11	63			15	64			9
6	48	88			43	58			6
3	—	81			38	38			3
0	—	—			—4	18			0
-3	58	—			-6	23			-3
HEARING LOSS					HEARING LOSS				

CASE 50.



Otitis media, bilateral, for at least 3 years and 5 months, more severe on the right.

Age at onset, 4 years 8 months.

Tympanic membranes were each incised three times.

No aural infection during the past 6 years.

Tympanic membrane at present on the right is retracted and shows a dry perforation in the posterior inferior segment. The left is intact, not retracted and has a good light cone.

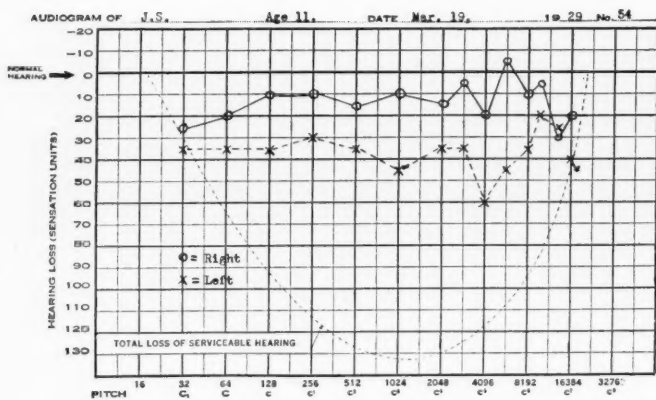
The 1A audiometer shows a loss for low tones in both ears and for high tones in the right.

The 4A audiometer shows slightly better hearing for voice in the left.

AC is greater than BC on both sides for the c2 fork.

X-rays: Clouding of both mastoids, more on the left.

CASE 50.

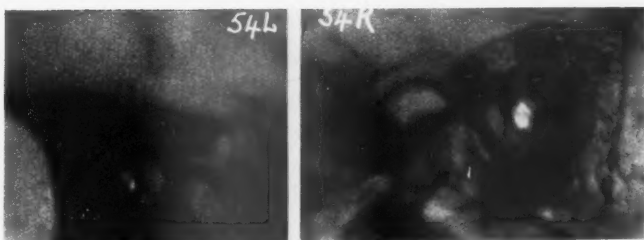


J.S. Age 11. Mar. 19, 1929

HEARING	RIGHT EAR				LEFT EAR				HEARING
LOSS	1	2	3	4	5	6	7	8	LOSS
30	54	56	85		48	55			30
27	41	68	83		XX	54			27
24	84	63	42		66	XX			24
21	51	14	18						21
18	18	34	61						18
15	XX	1X	XX						15
12	--	31	32						12
9	11	--	44						9
6	48	88	5-						6
3	XX	81							3
0	--								0
-3	51								-3
HEARING LOSS _____					HEARING LOSS _____				

CASE 54.





Otitis media, bilateral, for at least 2 years on the right and intermittently for  $10\frac{1}{2}$  years on the left.

Age at onset, 10 months.

Tympanic membranes were incised three times on each side.

No aural infection on the right during the past 4 years but the left has discharged intermittently to present time.

Tympanic membrane on the right at present is intact and the whole inferior segment posteriorly is calcified. There is a large perforation in the anterior inferior segment on the left through which a purulent discharge is seen.

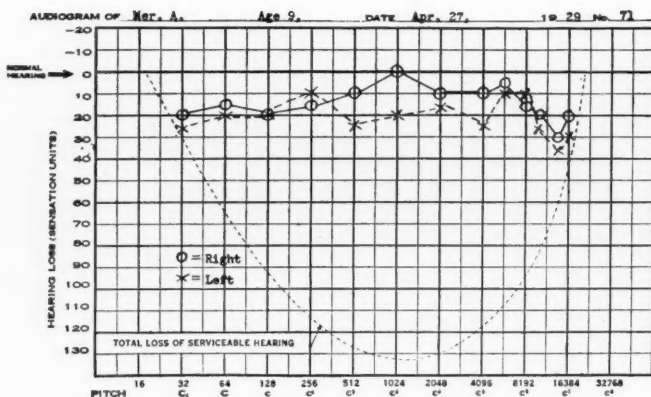
The 1A audiometer shows impairment for all tones on both sides, more marked on the left.

The phonographic audiometer shows 27 units loss for voice on the left and 3 units loss on the right.

BC is greater than AC on both sides for the c2 fork.

X-rays: Fairly good pneumatization of the right mastoid and dense sclerosis of the left.

#### CASE 54



Mer. A. Age 9. Apr. 27, 1929

HEARING LOSS	RIGHT EAR				LEFT EAR				HEARING LOSS
	1	2	3	4	5	6	7	8	
30	54	56			48	55			30
27	4-	68			23	54			27
24	84	63			66	88			24
21	51	14			41	42			21
18	18	34			88	23			18
15	23	12			68	85			15
12	22	31			—	—			12
9	11	63			—	44			9
6	48	88			48	58			6
3	—	—			—	38			3
0	—	—			—	—			0
-3	5-	—			—	—			-3
HEARING LOSS					HEARING LOSS				

CASE 71.



Otitis media, bilateral, for at least 5 years, more severe on the left.

Age at onset,  $3\frac{1}{2}$  months.

Tympanic membranes were incised once on each side.

No aural infection during the past  $3\frac{1}{2}$  years.

Tympanic membrane on the right at present is intact, not retracted, and has a good light cone. The left shows a dry perforation in the posterior inferior segment.

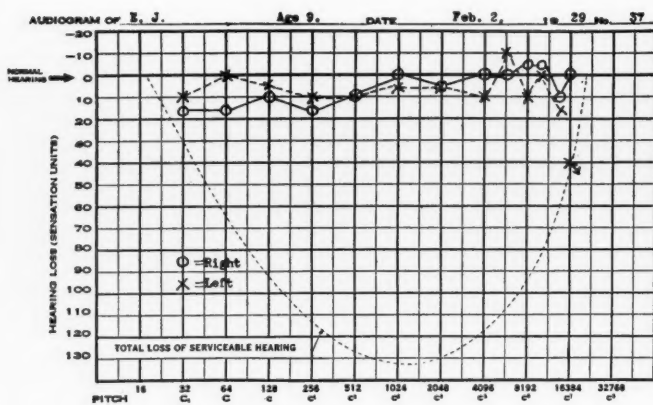
The 1A audiometer shows a slight impairment for high tones on the right and for all tones on the left.

The phonographic audiometer shows 3 units loss for voice on the right and slightly more on the left.

AC is greater than BC on both sides for the c2 fork.

X-rays: Both mastoids are well pneumatized but more extensively on the left.

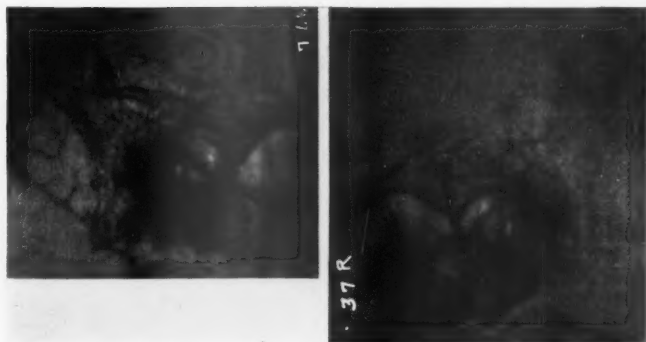
#### CASE 71.



E. J. Age 9 Feb. 2, 1929

HEARING Loss	RIGHT EAR				LEFT EAR				HEARING Loss
	1	2	3	4	5	6	7	8	
30	54	56			48	55			30
27	41	68			23	54			27
24	84	63			66	88			24
21	51	14			41	42			21
18	18	34			81	23			18
15	23	12			68	85			15
12	22	31			21	86			12
9	11	63			15	64			9
6	48	88			41	58			6
3	—	81			—	31			3
0	—	—			24				0
-3	—	—			—				-3
HEARING LOSS.....					HEARING LOSS.....				

CASE 37.



Otitis media, bilateral, for at least 12 days.

Age at onset, 13 months.

Tympanic membrane on the right was incised twice and on the left 4 times.

Has had occasional earache but no discharge during the past 8 years.

Tympanic membranes at present are intact, retracted and have good light cones. There is a healed perforation in the right.

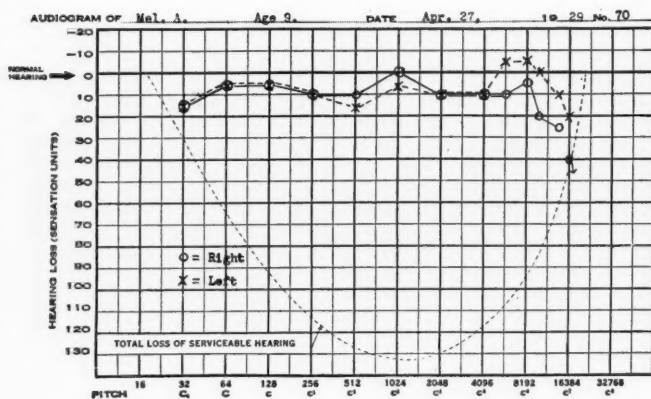
The 1A audiometer shows the hearing to be within normal limits.

The phonographic audiometer shows three units loss for voice on each side.

AC is greater than BC on both sides for the c2 fork.

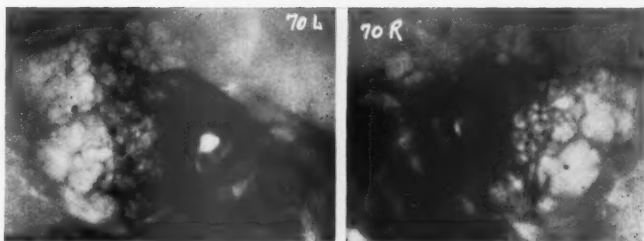
X-rays: Extensive pneumatization of both mastoids with some clouding of the right.

#### CASE 37.



Mel. A.		Age 9,				Apr. 27, 1929					
HEARING LOSS	RIGHT EAR				LEFT EAR				HEARING LOSS		
	1	2	3	4	5	6	7	8			
30	54	56			48	55			30		
27	41	68			23	54			27		
24	84	63			66	88			24		
21	51	14			41	42			21		
18	18	34			81	23			18		
15	23	12			68	85			15		
12	22	31			21	86			12		
9	11	63			15	64			9		
6	48	88			43	58			6		
3	38	81			38	38			3		
0	68	—			—	—			0		
-3	51	—			—	—			-3		
HEARING LOSS _____					HEARING LOSS _____						

CASE 70.



Otitis media, bilateral, for at least 5 years.

Age at onset,  $2\frac{1}{2}$  months.

Tympanic membrane on right was incised twice, and on left once.

No aural infection during the past 4 years.

Tympanic membranes are intact, not retracted and have normal light cones.

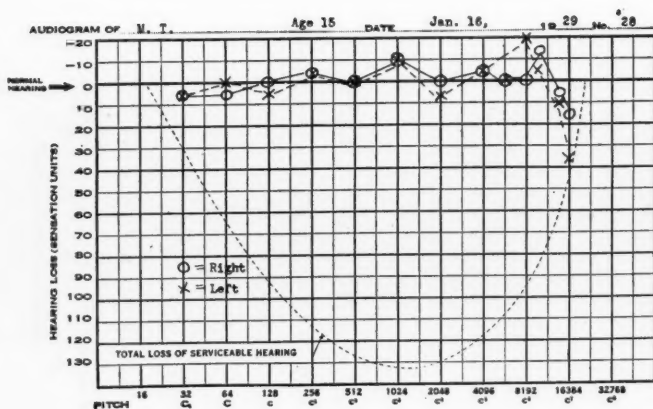
The 1A and 4A audiometers show approximately normal hearing.

AC is greater than BC on each side for c2 fork.

X-rays: Extensive pneumatization of both mastoids

CASE 70.

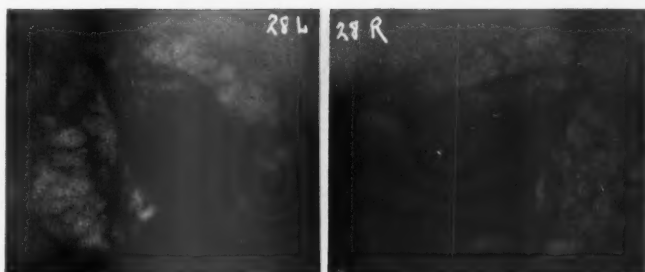




M. T.		Age 15.				Jan. 16, 1929			
HEARING	RIGHT EAR				LEFT EAR				HEARING
LOSS	1	2	3	4	5	6	7	8	LOSS
30	54	56			48	55			30
27	41	68			23	54			27
24	84	63			66	88			24
21	51	14			41	42			21
18	18	34			81	23			18
15	23	12			68	85			15
12	22	31			21	86			12
9	11	63			15	64			9
6	48	88			43	58			6
3	36	84			31	88			3
0	86	—			—4	18			0
-3	54	—			5—				-3
HEARING LOSS _____					HEARING LOSS _____				

CASE 28.





Otitis media, bilateral, for at least 1 year.

Age at onset, 13 months.

Tympanic membrane on the right incised twice and on the left three times.

No aural infection during past 12 years.

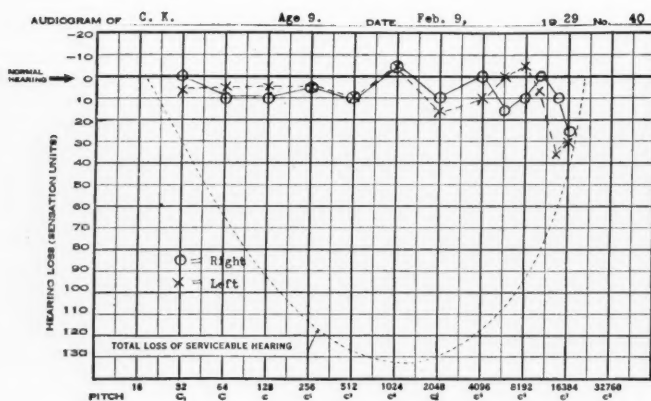
Tympanic membranes are intact, retracted, have good light cones but there is a healed perforation in the left.

1A and 4A audiometers show approximately normal hearing.

AC is greater than BC on each side for c2 fork.

X-rays: Extensive pneumatization of both mastoids.

CASE 28.

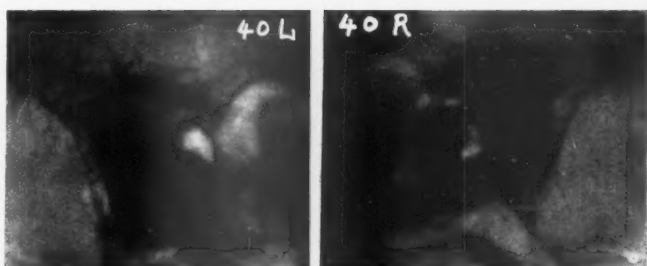


C. K. Age 9, Feb. 9, 1929

HEARING Loss	RIGHT EAR				LEFT EAR				HEARING Loss
	1	2	3	4	5	6	7	8	
30	54				48	55			30
27	41				23	54			27
24	84				66	88			24
21	51				41	42			21
18	18				81	23			18
15	23				—	85			15
12	22				11	86			12
9	11				15	14			9
6	48				43	54			6
3	31				31	13			3
0	66				48				0
-3	51								-3

HEARING LOSS \_\_\_\_\_ HEARING LOSS \_\_\_\_\_

CASE 40.



Otitis media, bilateral, for a least  $2\frac{3}{4}$  years.

Age at onset, 1 month.

Tympanic membrane on right was incised 7 times and on left 6 times.

No aural infection during the past 6 years.

Tympanic membranes are intact, retracted and have calcified plaques.

IA audiometer shows a slight loss for high tones.

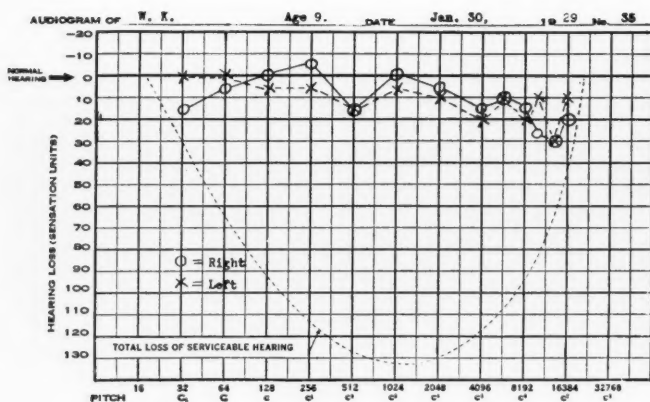
The phonographic audiometer shows slightly better hearing for voice on the right.

AC is greater than BC on each side for c2 fork.

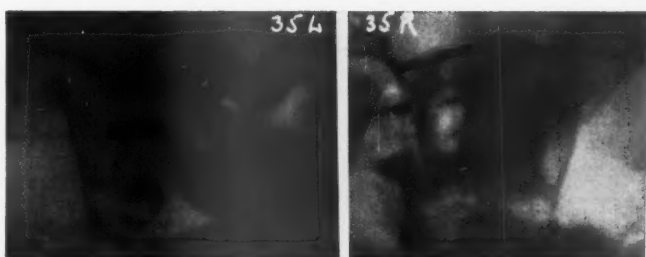
X-rays: Clouding of both mastoids, more on the right.

CASE 40.





W. K. Age 9		Jan. 30, 1929							
HEARING LOSS	RIGHT EAR				LEFT EAR				HEARING LOSS
	1	2	3	4	5	6	7	8	
30	54	56			48	55			30
27	41	68			23	54			27
24	84	63			66	88			24
21	51	14			41	42			21
18	18	34			31	23			18
15	23	12			68	85			15
12	22	31			21	86			12
9	11	63			15	61			9
6	48	88			48	58			6
3	38	81			31	88			3
0	8	68			81	18			0
-3	58	18			56	88			-3
HEARING LOSS					HEARING LOSS				



Otitis media, bilateral, for 10 months.

Age at onset, 6 months.

Tympanic membrane on the right was incised 4 times and on the left 2 times.

No aural infection during past 7 years.

Tympanic membranes are intact, retracted and atrophic with a calcified plaque anteriorly in the right.

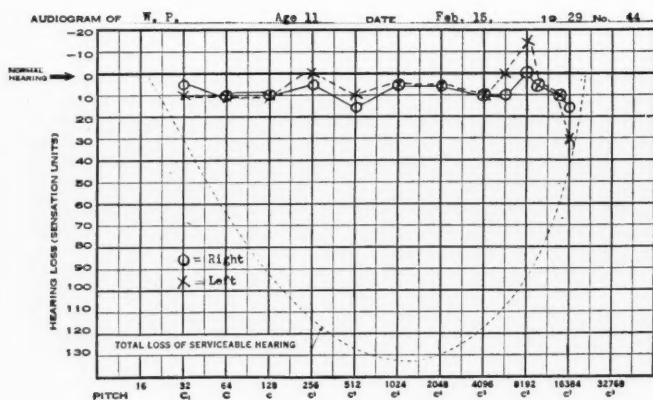
1A audiometer shows slight loss for high tones on each side.

The phonographic audiometer shows no loss for voice.

AC is greater than BC on each side for c2 fork.

X-rays: Dense sclerosis of both mastoids.

CASE 35.

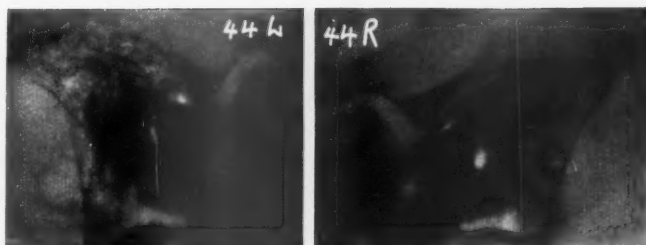


W. P. Age 11. Feb. 15, 1929

HEARING	RIGHT EAR				LEFT EAR				HEARING
LOSS	1	2	3	4	5	6	7	8	LOSS
30	54	56			48	55			30
27	41	68			23	54			27
24	84	63			66	88			24
21	51	14			61	42			21
18	18	34			81	23			18
15	23	12			68	85			15
12	22	31			21	86			12
9	11	63			15	64			9
6	48	88			43	58			6
3	38	81			31	34			3
0	64	—			—	14			0
-3	51	—			56				-3

HEARING LOSS

CASE 44.



Otitis media, bilateral, for at least 8 months.

Age at onset, 16 months.

Tympanic membranes incised once on each side.

No aural infection during past 10 years.

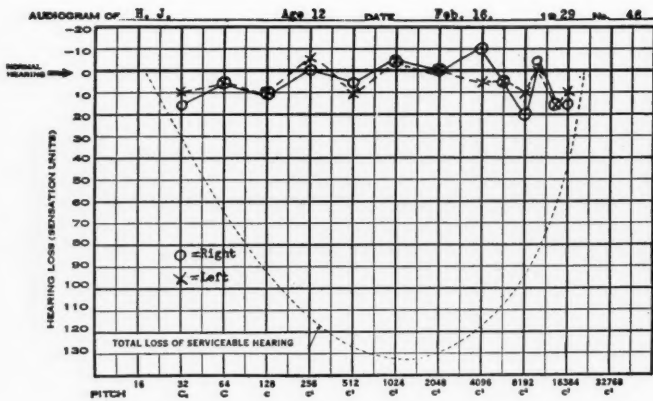
Tympanic membranes at present are intact, retracted and atrophic but have good light cones.

The 1A and 4A audiometers show approximately normal hearing.

AC is greater than BC on each side for c2 fork.

X-rays: Clouding of both mastoids, more on right than left.

#### CASE 44.

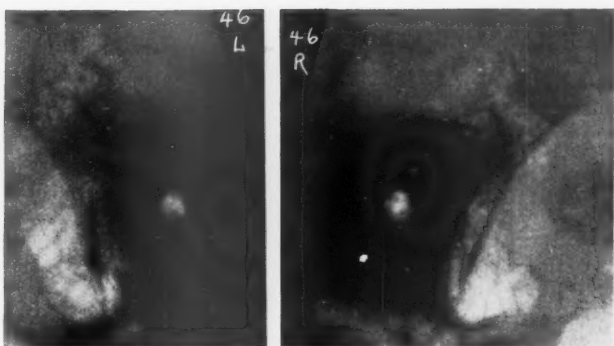


H. J. Age 12. Feb. 16, 1929

HEARING LOSS	RIGHT EAR				LEFT EAR				HEARING LOSS
	1	2	3	4	5	6	7	8	
30	54	56			48	55			30
27	41	68			23	54			27
24	84	63			66	88			24
21	51	14			41	42			21
18	18	34			81	23			18
15	23	12			68	85			15
12	22	31			21	86			12
9	11	63			15	64			9
6	48	8-			48	58			6
3	88	81			31	38			3
0	—	28			—	18			0
-3	51	—			56	—			-3
HEARING LOSS _____					HEARING LOSS _____				

CASE 46.





Otitis media, bilateral, for three weeks, more severe on the left.

Age at onset, 9 months.

Tympanic membrane on right incised twice and on left three times.

No aural infection during past 10 years

The tympanic membranes at present are intact, not retracted and have normal light cones.

The 1A and 4A audiometers show approximately normal hearing.

AC is greater than BC on each side for c2 fork.

X-rays: Extensive pneumatization of both mastoids.

CASE 46.

## II.

### PNEUMOCELE OF THE FRONTAL SINUS, WITH REPORT OF A CASE, PROBABLY SECOND- ARY TO BLASTOMYCOSIS.\*

BY SAMUEL IGLAUER, M. D.,

CINCINNATI.

In his interesting Commentaries on Surgery, published in 1855, Guthrie<sup>1</sup> made the following observation:

"After a wound of the frontal sinus has healed, the air has been known to raise up the integuments of the forehead into an elastic, crepitating swelling whenever the patient blew his nose, so that a compress and bandage were required for its relief; but these cases are very rare."

In these few words Guthrie described the origin of one variety of traumatic air sacs of the frontal sinus. In 1867 Warren<sup>2</sup> of Boston reported his classic case in a patient presenting a bilateral swelling on the forehead. The swelling contained air and pus, and at operation necrosis of the anterior plate of the frontal bone was found. There was a history of trauma in this case also.

In 1891 Von Helly<sup>3</sup> collected the histories of eight cases from the literature (including those of Guthrie and Warren) and added one of his own.

Since 1891, Levinger,<sup>4</sup> Rosenberg,<sup>5</sup> Benjmains,<sup>6</sup> Boenninghaus,<sup>7</sup> Grünwald<sup>8</sup> and Ferreri<sup>9</sup> have each reported a single case, while Benjamains collected three additional cases from the literature, giving a total of eighteen cases up to the present time.

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\*Read before the Middle Section Meeting of the American Laryngological, Rhinological and Otological Society, at Rochester, Minn., January 21, 1930. From the Department of Otolaryngology, Cincinnati General Hospital, University of Cincinnati, College of Medicine.

## PATHOLOGY.

In order that air, under pressure, may escape from the frontal sinus and produce an aerocele adjacent to the sinus, there must be a defect in the bony wall.

If the tumor appears on the forehead, it gives rise to an external pneumocele or pneumatocoele. If the defect is in the posterior wall, it may give rise to an internal or intracranial pneumocele. The latter usually follows a fracture in the posterior wall of the frontal sinus, with a tear in the dura, through which the air may be forced into the meninges, the frontal lobe of the brain, and even into the lateral ventricles. (Dandy.<sup>10</sup>) The rhinologist is especially concerned with external pneumocele, while the intracranial form is of greater interest to the brain surgeon.

In an external pneumocele the defect in the sinus wall may be due to trauma or operation, or, as pointed out by Von Helly,<sup>3</sup> a congenital cleft or dehiscence may be present.

The defect may also result from caries or necrosis of the bone due to syphilis, osteomyelitis or sinusitis. In the case which I am about to report it seemed certain that blastomycosis involving the frontal bone was the cause of the lesion.

After a recent fracture involving the frontal sinus, air may collect over the sinus in the form of a subcutaneous emphysema which may spread widely in the scalp, but which, however, does not constitute a true pneumocele.

For the production of a true pneumocele the mucous membrane or the periosteum, or both, must be intact over the bony defect, so that when the patient sneezes, blows his nose or makes forced expiration with the mouth and nostrils closed, the air will cause a ballooning of the mucosa or an elevation of the periosteum in the form of an air sac. With the subsidence of the air pressure the sac tends to collapse and may be emptied upon the application of external compression.

A rare form of pneumocele is the one that occurs in connection with a mucocoele. In this case there is no defect in the bone, but the sinus walls are thinned and the cavity dilated, and, with the escape of the fluid contents into the nose, air enters to take its place. Boenninghaus<sup>7</sup> was able to follow the development of such a case over a period of nine years.

When the mucocele finally discharged its contents, the dull percussion note previously present was supplanted by a tympanitic note, and upon operation no fluid was found in the frontal sinus.

It is rather remarkable that external operations on the frontal sinus are not more frequently followed by pneumocele, but the comparative rarity of this complication must be due to the firm scar tissue and the adhesions of the periosteum and scalp to the margins of the operative opening.

#### REPORT OF CASE.

The following case, which came under my observation in June, 1928, presents some unusual features:

History: The patient, a man, aged 43, was admitted to the Cincinnati General Hospital on May 31, 1928. He had served in the U. S. Army (July, 1918 to February, 1919), from which he had been discharged free from physical defects. He gave a history of a syphilitic infection thirteen years before his admission to the hospital. While in the army he had received a thorough course of antiluetic treatment, and in the spring of 1928 he received a second course of treatments. His chief complaints were (1) aching all over the body and of numerous tender swellings in different areas of the body, (2) drowsiness. His recent illness began in January, 1928, and he had been in another hospital for the preceding two months.

On admission he stated that he had had persistent headache for five months. There was a soft, fluctuating, tender, plum-sized mass in the frontal region above the bridge of the nose. There was a similar larger swelling at the sternal end of the right clavicle. There was also a visible and palpable swelling in the course of the seventh rib in the anterior axillary line. There was a discharging sinus just above the os calcis on the left. All of these swellings (except the frontal) gradually increased in size, fluctuated and filled with pus, which was either aspirated or discharged through fistulous tracts. The underlying bone seemed to be involved in all these abscesses. He also developed a mediastinal abscess.

Laboratory and Clinical Tests.—Repeated Wassermann tests were all negative—i. e., two blood tests, one spinal fluid test

and one provocative test. Red blood cells, 3,980,000; white blood cells, 10,200.

Acid-fast bacilli were absent in repeated examinations of the sputum and pus from the abscesses. Guinea pig inoculation proved negative.

Stereoscopic X-ray examination of the skull made in the lateral position and also in the posteroanterior position showed no findings which would indicate bone pathology.

Neosalvarsan and potassium iodid were administered without any permanent therapeutic effect.

A biopsy was made on a bit of tissue removed from the sinus on the left heel.

Pathologic report (August 2), 1928: ". . . There is a well defined granuloma, with many giant cells; these, in turn, contain well developed blastomyces in all stages of growth." (Dr. N. C. Foot.)

Diagnosis: Blastomycosis. (Fig. 1.)

The final conclusions, recommendations and summary were as follows:

"This patient was admitted to the hospital with a tentative diagnosis of tertiary syphilis. He has been in the hospital since May 31 to this date (July 18), partially due to inability to determine the nature and cause of his disability and partly due to the seriousness of his physical condition. The staff for a number of weeks felt that the condition was either tuberculosis or tertiary syphilis, but did not make a definite statement as to which it was, as it did not present typical symptoms of either condition. A definite diagnosis was made only after report of biopsy (July 17), which definitely indicated that the patient was suffering from a blastomycosis. The pathologic report of the second biopsy completely confirmed the diagnosis of blastomycosis. Recommendations: Treatment should be symptomatic and palliative. Prognosis poor."

#### PNEUMOCELE.

The swelling on the patient's forehead appeared about two months before we saw him, while he was in another hospital.

On June 1, 1928, Dr. Roger Morris made the following note: "There is a swelling in the frontal region in the midline meas-

uring  $3\frac{1}{2}$  cm. in diameter, elevated about 1 cm. The swelling is tender, fluctuates and is apparently causing a necrosis of the frontal bone."

Two weeks later (June 18) I noted the following:

"In the midline, about one inch above the glabella, there is a depression in the frontal bone which admits the tip of the ring finger. There seems to be a bone defect present. This area is slightly tender and gives a crackling sensation on palpation. When the patient holds his nose shut and blows, a swelling about the size of a plum appears at the site of the depression. (Fig. II.) Pressure upon this swelling causes the air which it contains to disappear with a blowing noise in the nose. In the left nostril anteriorly there are clots of blood. There is a varix on the left side of the septum anteriorly. There is no purulent secretion in the nose."

Impression.—Defect in the frontal bone with a fistula connecting with the nasal air passages, probably through the frontal sinus, on one side or the other, through which the patient can force air. This lesion is probably of luetic origin.

Since the X-ray examination had failed to reveal the nature of the lesion in the frontal bone, the following procedure was adopted:

A small amount of novocain solution was injected into the skin covering the swelling on the forehead. A needle was then introduced into the tumor, keeping parallel to the surface, and about 4 cc. of lipiodol was injected. Bare bone could be felt against the tip of the needle. The patient stated that he could taste the lipiodol and felt it in his pharynx.

X-rays were then immediately taken in frontal and lateral views. The frontal view of the X-ray film showed the right frontal sinus almost completely filled with lipiodol, and a streak of lipiodol could be seen in the region of the nasofrontal duct with the escape of some of the lipiodol into the right nasal chamber. On the lateral view a small fistulous tract could be seen leading into the frontal sinus, which was almost completely filled with lipiodol. The X-ray demonstrated the air connection between the nose, the frontal sinus and the pocket under the scalp. Fig. III.

Twelve days after the lipiodol injection, when the patient inflated his nose, the pneumocele was only about half the size

of the original tumor. Six days later he was unable to inflate the pneumatocele, probably because of his enfeeblement.

On July 19, 1928, the patient was transferred to another hospital, where he died on August 10, 1928. No autopsy was performed.

Case 2.—Male, aged 32. About one year later a second case of disseminated blastomycosis was admitted to the hospital. This case is cited because the frontal bone was involved as well as the body of a vertebra, a rib and one scapula. X-ray examination of the frontal bone showed that both the inner and outer table were completely destroyed. Although this lesion was near the frontal sinus, the patient was unable to inflate the firm depressions by blowing his nose, there apparently being no communication with the upper air passages. The diagnosis in this case was made at biopsy and was confirmed by autopsy.

Diagnosis.—The diagnosis of pneumocele is made from the history and from the presence of an expanding (during forced expiration) and disappearing tumor on the forehead. It is elastic, cushionlike and compressible on palpation, and may disappear on pressure. Percussion should elicit a tympanitic note. X-ray examination usually shows a translucence of the tumor and may reveal the bone defect. The injection of lipiodol, as demonstrated in my case, renders the diagnosis more certain. These combined characteristics should serve to differentiate it from other swellings in this region, namely, a periostitis, an osteomyelitis, a neoplasm, a meningocele, a pyocele, or mucocele. An external frontal sinus operation is usually necessary for the cure of a pneumocele.

#### COMMENT AND SUMMARY.

Two cases of disseminated blastomycosis with multiple bone involvement are cited. In each case there was a lesion in the frontal bone. In case I it was necessary by repeated laboratory tests to exclude both syphilis and tuberculosis as a cause of the bone destruction. The biopsy findings warranted the conclusion that the bone lesions were due to blastomycosis, although the tissue was not removed from the frontal region. This case presented an unusual opportunity of observing a

case of pneumocele from its incipency until it had become fully developed.

The usual X-ray technic failed to show the defect in the frontal sinus wall and the nasal connection of the pneumocele.

The configuration and relations of the pneumocele were clearly demonstrated after the injection of lipiodol into the pneumocele. The injection of lipiodol constitutes a distinct aid in the diagnosis of these unusual air tumors.

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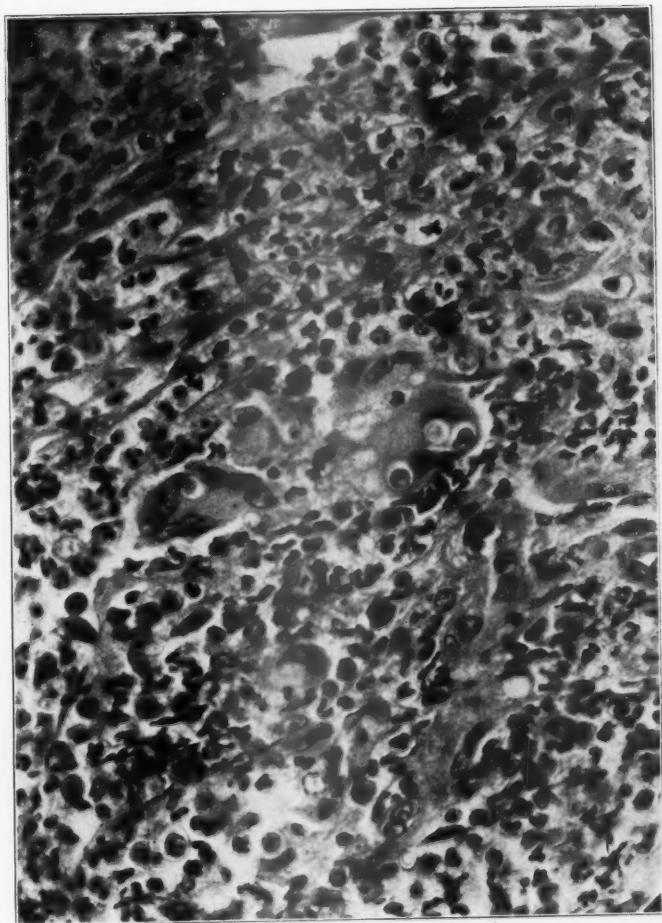


Fig. 1. Section of tissue showing numerous giant cells which contain well developed blastomyces in all stages of growth.



Fig. 2. Showing the patient inflating the pneumocele.



Fig. 3. Lateral view showing the pneumocele and the frontal sinus almost completely filled with lipiodol which can be seen trickling into the nose through the nasofrontal duct.

### III.

#### THE PRESENT FINDINGS IN A CASE OF ACROMEGALIA OPERATED ON BY THE HIRSCH METHOD SEVENTEEN YEARS AGO.

BY W. E. SAUER, M. D.,

ST. LOUIS.

This case was reported and published in the *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, December, 1913. I will, therefore, only briefly outline the past history.

Mr. S. D., Lithuanian, now aged forty-nine, had been normal in growth and development, and enjoyed good health until the age of seventeen. At that time he began to have frontal headaches, which usually came on in the morning. He was, however, not incapacitated until the age of twenty-two, when he noticed a considerable enlargement of the hands, feet and lower jaw. His vision was becoming impaired and he became so weak generally that he was forced to give up his occupation as a coal miner. He tried other occupations, among them that of bartender, but owing to the large and clumsy hands this was also given up.

For the next ten years his symptoms gradually increased. The headaches became more severe, his vision more and more impaired. His muscular weakness also progressed to the extent that a walk of a block became a hardship. The overgrowth of his extremities and face became more noticeable. In 1911 he came under the observation of Dr. L. H. Behrens of St. Louis, who reported his findings as follows:

"At the time I first saw him he was thirty-two years of age. He had the typical acromegalic facies. (Fig. 1.) The teeth showed marked separation and his tongue was large. The hands were spadelike and immense. The greatest circumference of his thumb was three and one-half inches, of his hand ten and one-half inches. His feet were broad and he wore No. 11 shoes, double E width. His sight was greatly impaired in the left eye. There was a definite beginning atrophy of the left optic nerve. His vision was 15/200 in the left and 15/12 in

the right eye. The visual fields were contracted as shown in Fig. 2. The X-ray of the skull showed a very large cella turcica with thin walls."

He was observed for a year without treatment. His sight became progressively worse, his weakness increased and his headaches persisted. At this time he was also seen by Dr. Ernest Sachs at the Barnes Hospital, who expressed the opinion that the disease was spreading and that an operation was distinctly indicated. The question of the operative procedure was carefully considered. About this time Hirsch had reported twenty-six cases which he had operated on by his endonasal method with a mortality of only three. These results were the best reported up to that time. The Hirsch operation was decided upon and the patient was sent to the hospital on November 28, 1912, and on the following day, after the usual preparations for a submucous resection of the septum, he was sent to the operating room. The septum and the anterior wall of the sphenoid was carefully cocaineized with a 20 per cent solution of cocain and in addition a one-half of 1 per cent solution of cocain was now injected on each side of the septum near its posterior end. The middle turbinate on the left side was removed, after which a submucous resection was done in the usual way. The mucous membrane on the anterior wall of the sphenoid was elevated and pushed to the side until the natural openings of the sphenoid were reached. The nasal septum was removed entirely, the openings of the sphenoid enlarged and the partition between the two sphenoid cavities was removed. Owing to the large size of the skull, with unusually large sphenoid cavities, as shown in Fig. 3, there was no difficulty in obtaining a good view into the interior of these cavities. The upper wall of the sphenoid was found to be bulging downward, as shown in Fig. 3. By means of a long, slender chisel an opening was made in the middle line, and this opening was enlarged with a punch forceps. The bone was not as thin as I had expected to find it, but there was no difficulty in securing an opening about one centimeter long and three-fourths centimeter wide. The dura was incised by means of a sickle shaped knife.

At this stage the patient became unruly and nothing further was done. The mucous membrane covering the anterior wall

of the sphenoid was torn to such an extent that I packed the nose in the usual way and did not place a drain between the septal mucous membranes as recommended by Hirsch at that time. Hirsch now packs the nose as is done in a septum operation, but he enlarges the opening in the mucous membrane leading into one or the other sphenoid cavity, securing drainage in this way. The pack was removed at the end of twenty-four hours; there was no unusual bleeding or any other unusual symptoms following the operation. The patient noticed a marked difference in his heart action and stated that he felt "much lighter"; he particularly noticed that the foul odor he had previously complained of had disappeared. He was discharged from the hospital in ten days, at which time an ocular examination by Dr. Parker showed the visual acuity in the right eye normal, left 17/120.

December 10, 1912, his vision in the left eye was 17/60.

Eleven months after the operation the visual acuity in the right eye was normal. The left eye was 17/40, and the visual fields were definitely wider. He had in the meantime gone to work as a laborer, unloading ice and other ordinary jobs without any discomfort. He was able to wear smaller hats and smaller shoes than he could prior to the operation.

He was not seen again until October, 1929, when he returned to St. Louis because he was out of a job. Dr. Behrens requested him to go to the Barnes Hospital for observation. He had remained very well during the intervening years; had been free from headaches. His eyes had not given him any trouble and he had been working on a farm ever since he left St. Louis. His appearance is still strikingly acromegalic, as shown by Fig. 4. His thumbs measured three and one-half inches in circumference, but the circumference of his hands was only nine and one-half inches compared to ten and one-half inches before the operation. He was wearing a No. 10 shoe, whereas he wore a No. 11 prior to the operation. The X-ray of the skull shows a definitely enlarged sella (Fig. 5), but in the opinion of the roentgenologist, Dr. Sherwood Moore, the sella appeared smaller than seventeen years ago. The vision in the right eye was 20/30 and the left eye 20/50. The visual fields show an improvement, as shown in Fig. 6.

In this case a simple decompression not only saved the vision in the left eye but there was some regression of the acromegalic symptoms. Certainly after seventeen years we can conclude that the progress has been permanently arrested.

Surgery of the hypophysis has made great strides when we consider that the first operation on the living was done by Horsley in 1904. Frazier (*Annals of Surgery*, July, 1928) states that the surgery of the pituitary lesions has grown during the past fifteen years from a problem of minor to one of major importance. The first pituitary operation in his neurosurgical clinic was performed in 1912, and today pituitary lesions represent 15 per cent of their register of intracranial tumors. The question as to whether the intracranial route or the transsphenoidal offers the better route of attack is apparently not settled. Frazier states that after wavering at different periods between one and the other, they have in the past three years abandoned the transsphenoidal route. Although realizing with the latter a lower operative hazard, they were forced to recognize its limitations, since recurrences of symptoms were not infrequent. In a recent series of "eleven transfrontal craniotomies" there was one fatality. In his series of thirty-five consecutive transsphenoidal operations the mortality was only  $3\frac{1}{2}$  per cent.

Fedor Krause (*Deutsche Medizinische Wochenschrift*, April 22, 1927), on the other hand, strongly favors the transsphenoidal route because of the lessened hazard and the gratifying results he has obtained. He believes that the transfrontal route should be reserved for those cases where the tumors are known to be suprasellar and of large size and when the transsphenoidal route has not been successful. He follows the Schloffer method of temporarily resecting the external nose and removing the turbinates. Krause reports a case of acromegalia in an army officer in which the hands were much enlarged but not the feet. The headaches were intense and he had frequent attacks of vertigo. There were no eye symptoms. The sella showed a distinct enlargement. On August 1, 1926, an operation was performed by the transsphenoidal route. On opening the sella a gray, reddish tumor presented itself. Palpation with the finger revealed a pulsating tumor which was synchronous with the heart beat but a definite artery could

not be felt. As he gently tore open the capsule in the midline a terrific arterial hemorrhage followed, which was controlled by packing the wound with gauze. This pack was allowed to remain twenty-one days before he dared remove it. The patient left the hospital sixteen days later without any further trouble. Five months later, much to his surprise, all symptoms had disappeared and there was a marked regression of the enlargement of his hands. When seen, eleven months after the operation, the good results were still maintained and there was further improvement in the acromegalic symptoms. This case again illustrates the results that may be obtained from a simple sellar decompression. According to Hirsch, Von Eisilsbergs has had the largest experience with the Schloffer method, viz., twenty-six cases with a mortality of six, approximately 23 per cent. Hirsch has a total of 113 operations on 81 cases, with 14 deaths, a percentage of 12.4. He explains that a number of cases, owing to the recurrence of symptoms, were reoperated on, sometimes months, sometimes years apart. Two of his cases were diabetics, one of which was operated on while in coma. Two cases died, six to seven weeks after the operation, from meningitis. Four of his cases were secondary operations. In the solid tumors, when recurrences had taken place and secondary operations were attempted all died. For several years he abandoned secondary operations on solid tumors, but now uses radium with good results. He attempts to explain this on the ground that it is impossible to separate the septal mucous membranes as in the primary cases. The operative field communicating with the nasal cavities cannot be as favorable in these cases. He did not lose a single case when a secondary operation was done on cystic tumors. Hirsch believes that meningitis results from tumor rests which remain after curetting and have become infected, an abscess forms which when confined to the tumor and its vicinity may be relieved by aspiration, at least he was able to do so in two of his cases. He separates the septal mucous membranes and introduces a glass tube into the sella against the tumor and aspirates the accumulated fluids by means of suction. Hirsch reports that in his last twenty-two operative cases he has not had a single fatality.

In order to prevent recurrences in the cystic cases Hirsch places a drainage tube in the cyst at the time of the operation



and allows it to remain for fourteen to twenty-one days. If it should become occluded he changes the drain. If an opening as large as a grain of rice remains this is sufficient for permanent drainage.

Cushing's sublabial method differs from Hirsch's method only in that an incision is made under the upper lip and the nasal septum removed, elevating the mucous membrane through this incision. The operation is done under general anesthesia. In some cases a preliminary tracheotomy is done. Cushing's has a mortality record of 13.2 per cent in 200 operations done by this method. The majority of Cushing's operations have been done by this method. In his last fifty consecutive cases he has had only one death, reducing his total mortality to 7 per cent.

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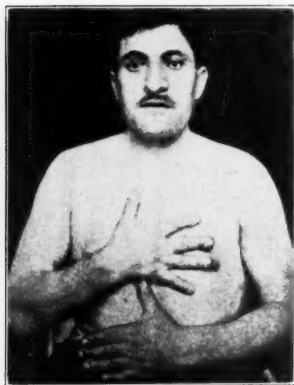


Fig. 1.

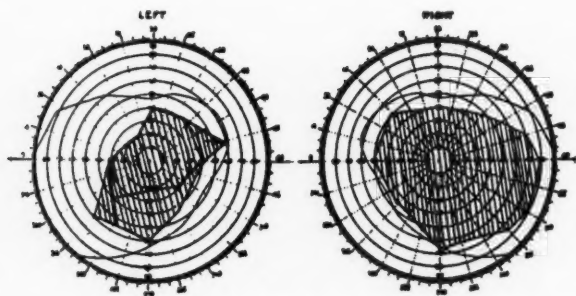


Fig. 2.



Fig. 3.



Fig. 4.



Fig. 5.

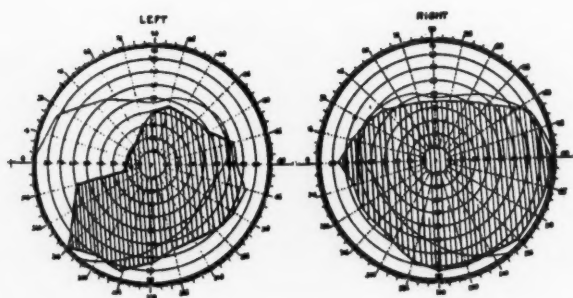


Fig. 6.

#### IV.

### MASTOIDITIS WITH GASTROINTESTINAL DISTURBANCE IN INFANTS.

BY JOHN W. CARMACK, M. D.,\*

INDIANAPOLIS.

In 1921 Maurice Renaud of Paris, France, reported having found pus at postmortem in infants who had died of cholera infantum. Later, Byfield and Dean, then at Iowa University, called attention to the presence of pus not only in the mastoid spaces but in the nasal sinuses of children dying of gastroenteritis.

In 1923 the staff of the St. Louis Children's Hospital presented a similar report, and in 1925 a further report of 15 infants under two years of age with gastrointestinal symptoms upon whom they had done mastoid operations, with eight recoveries and seven deaths.

Later still, a report of 30 cases of marked gastroenteritis upon whom mastoid operations had been done during 1926 and 1927, with twenty-two recoveries and eight deaths. These reports stimulated considerable investigation and activity throughout the country, with varying opinions and results.

There has been some question as to whether mastoiditis is the cause or a coincidental infection; at any rate, as to its exact rôle in the etiology of these gastrointestinal disturbances in infants. It is not difficult to see why there might be confusion when we consider the difference in the effect of pathogenic organisms in different localities. There has occurred also a decided change in the effect of certain infections in the same locality over a period of a few years. For example, the so-called "flu" infection, which was largely respiratory for a time, now is frequently complicated by gastrointestinal symptoms, resulting in so-called intestinal flu.

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There have been some conflicting opinions also as to the advisability of external mastoid drainage and the proper time for interference in these infants, because of unsatisfactory results. This is not strange, however, when one considers the difficulty in diagnosis, the selection of the time for operative interference, and the intense preoperative and postoperative attention which is required for a successful outcome.

It appears to us that there are two rather well-defined types of cases in which mastoid and gastrointestinal disturbances occur. The first, in which we find an acute infection in the respiratory tract, including the middle ear and mastoid, and at the same time or immediately following, a violent enteritis producing frequent stools, sometimes bloody, rapid loss of weight, dehydration and toxemia. The second type, in which mastoid infection is present but it is doubtful whether the gastrointestinal disturbance is due to actual infection of the bowel or to a toxic disturbance, is characterized by less violent symptomatology, such as intermittent vomiting, inability to gain weight, or an intermittent gain and loss of weight, with very little if any diarrhea.

This report is based upon 28 infants, under two years of age, who were operated on for mastoiditis and who had gastrointestinal disturbances varying from inability to take food, intermittent vomiting and loss of weight, to violent diarrhea with anhydremia and temperature of 101 to 105 degrees with extreme toxemia. Twenty of these cases recovered and eight died. The first six were operated on during the active stage of an dehydrating diarrhea with high temperature. The mastoid spaces of these six were filled with pus and very thick edematous mucosa. Of these six, three died and three recovered after a stormy convalescence. Our most unsatisfactory results have been in cases operated during this stage. In twenty-one cases both mastoids were operated at the same time. Of the seven operated upon on one side, it was necessary to operate upon the other side later in four. Nineteen of the twenty-eight contained swollen mucosa, pus, granulation tissue and debris. The other nine were of the sclerotic type.

The fact is definitely established that mastoiditis is present in certain of these gastrointestinal disturbances in infants, but it appears there is much to be desired in the standardization

of our ideas regarding the selection of the time for operation and the preoperative and postoperative treatment. There can hardly be another condition where individual attention and decisions are more necessary, but there are certain procedures which we have found must be observed to obtain uniformly good results. The diagnosis is often difficult and cannot be intelligently made except by the closest cooperation of the pediatrician, the otolaryngologist and the roentgenologist. In the first place, there must be an elimination or conformation of all other conditions, mechanical or infectious, which could produce trouble of this kind, such as chest, heart, urinary or primary gastrointestinal disturbances; and, just as important, there must be demonstrable evidence of disease in the mastoid before surgical intervention is justifiable.

The appearance of the drum membrane is not always a safe indication of the pathology inside. It has been our observation that few if any of these infants with marked gastrointestinal disturbance have a normal drum membrane, especially where there is considerable dehydration. If the drum is intact it usually presents a gray, lusterless appearance, with some or all of the normal landmarks obliterated. If there is bulging of the upper posterior portion of the drum membrane it is a valuable aid, but in a few cases drum incision has revealed a dry air space in the middle ear, with positive culture, while the mastoid space was filled with pus which did not escape through the aditus. In these cases drainage had apparently continued through the eustachian tube. Fortunately, however, most of these infants with mastoiditis either present a bulging drum, give a history of a discharging ear following an upper respiratory infection, or show X-ray evidence of disease. Objective indications over the mastoid area externally have been notably absent.

The X-ray has been of considerable value in diagnosis, but the plates are difficult to interpret and may be misleading. Their possible value depends upon acquiring as perfect a mental image of the normal infant mastoid as possible and a comparison with that. Our X-ray studies of the normal infant mastoid lead us to believe the space to be larger than has been indicated between the first and second year of life. We have also observed cell formation, both by X-ray and at

operation, very frequently as early as one year, whereas we are taught it occurs at two years or after. The X-ray picture of the normal infant mastoid is rarely a clean cut, well outlined space, because of the lack of bone density in the infant skull, the shallowness of the air space and the possible presence of more or less embryonal tissue which has not been absorbed. (Fig. 1.)

When there is X-ray evidence of mastoid pathology we have noted two rather distinct variations from the normal. In the earlier or more active type, where inflammatory swelling and pus fills the cavity, the plate gives a generalized increased density compared to the normal. (Fig. 2.) In cases of longer standing, when there has been suppuration with necrosis of the mastoid content and discharge through the ear, the cavity may be quite transparent to the X-ray, with a handlike area of unusual density surrounding. (Fig. 3.) Bone necrosis, while not always shown by the X-ray, has been found repeatedly at operation. In this latter so-called sclerotic type we have found a free cavity the lining of which was a moist necrotic wall of bone and underneath a bony wall of increased density. In two of this group a large area of the sinus plate was destroyed and in one the dural plate.

The selection of the time for operation is of major importance. Our most unsatisfactory results have been in cases that were operated upon during the height of an acute bowel inflammation with frequent stools, high temperature, marked toxemia and dehydration, regardless of preoperative preparation. It would seem to us advisable, if possible, to delay operation until the storm subsides, as there is always more or less shock and febrile reaction after mastoid surgery in these infants. Preoperative attention has been directed towards restoring the body fluids to as nearly a normal state as possible. Salines or Ringer's solution intravenously, subcutaneously and intraperitoneally have invariably been used, but we feel that blood transfusion is indicated where the hemoglobin is under 70 or the red cell count is under 4,000,000. All operations have been done under novocain anesthesia, with a preliminary dose of  $\frac{1}{8}$  to  $\frac{1}{2}$  grain of codein, performed as rapidly as is consistent with careful surgery and with as little loss of blood as possible, the aim being to remove all necrotic tissue and



provide free drainage from the mastoid antrum and middle ear. The wound has been partially closed in some cases, and occasionally the skin incision heals rapidly but most have required reopening. It is our impression that the safer plan is to keep all wounds wide open until the mastoid cavity heals by granulation.

The postoperative care has been supportive and stimulating. Fluids in whatever manner are considered advisable; food in as rapidly increasing quantities as will be tolerated. Repeated blood transfusions, if necessary, or whatever other skilled pediatric attention may be indicated. Local attention to the wound has consisted of daily dressings with free drainage of first importance. The skin surface and back of the head and neck must be kept clean to prevent skin abscesses and boils which occur and are extremely annoying and dangerous.

I wish to report one case in which there was middle ear and mastoid infection present for at least ten months, with spitting of food and intermittent loss and gain of weight, which finally developed evidence of acute intestinal infection with diarrhea, vomiting and anhydremia.

#### CASE REPORT.

First Admission.—Patient was admitted May 28, 1929, to the James Whitcomb Riley Hospital for Children; age five months; weight 12-10. Examination showed the following history:

Full term, normal delivery; breast-fed. No history of contagious diseases. At two weeks took cold. Both ears discharged for several weeks and stopped. The ear discharge has recurred intermittently since, with vomiting and loss of weight following each exacerbation. The baby had not taken its food well and spit up much of it since two weeks of age. There was no tendency to diarrhea but it had gained in weight very slowly.

Examination: Poorly nourished; marked obstruction to breathing; both ears discharging pus. X-ray of thymus, negative. Chest plates showed peribronchial thickening. Serology, negative. Temperature, 99 to 100 degrees. Urinary tract, normal. Gastrointestinal tract, apparently normal. Tonsils were

sufficiently large to be obstructive; the adenoid completely obstructed the nasopharynx. The child was placed on breast feeding supplemented by lactic acid milk. Spitting of food and weight loss continued. Mastoids were X-rayed and showed slight cloudiness. Removal of the tonsils and adenoid was advised and this was done one week later. Recovery from this was uneventful, breathing became free, the ear discharge stopped, the feedings were retained better, and the patient was discharged in three weeks.

For three months there was a gradual improvement. Weight at this time,  $14\frac{1}{2}$  pounds, when a severe diarrhea began. The baby was returned to the outpatient department, and at that time a diagnosis of toxic diarrhea and anhydremia was made. A formula was prescribed and after two weeks the diarrhea had stopped, but there occurred a swelling at the base of the skull under the left mastoid area which suppurated and was incised. This was diagnosed as a suppurative lymph gland. The wound healed rapidly, temperature returned to normal and the baby began slowly to gain weight. This continued for about two months more, when the child was admitted to the Riley Hospital with a severe diarrhea, ten to fifteen stools daily, some vomiting, temperature of 103 to 104 degrees, extreme dehydration and toxemia and a rapid loss of weight. General examination was otherwise negative. Ears were dry but the left drum membrane was thickened and lusterless. There was a small perforation in the upper half. Re-X-ray of the mastoid was done. The right mastoid area contained air and there were visible septa, with no evidence of bone destruction or thickening. The left mastoid contained air, but there was no evidence of cellular septa. This mastoid area was surrounded by a definite line of dense bone, indicating an inflammatory change. Operation on the left mastoid was advised when the general condition permitted. Operation was done ten days after admission, under novocain anesthesia. On removing the bone cortex a cavity was found lined with grayish necrotic bone, beneath which was an increased density of the bone. Culture from the mastoid showed a small cocci in pure culture which at first was grayish white in color, but after 72 hours became an aureus. This organism was decidedly hemolytic. The mastoid wound healed promptly, temperature sub-

sided in ten days, the vomiting subsided, and the feedings were gradually increased. This child gained five pounds in weight during the next four weeks, when it was discharged, and has had no further trouble. (Fig. 4.)

The outstanding features of this case are:

First, the fact that following a cold at two weeks otitis media occurred and continued with intermittent exacerbations for ten months, each exacerbation being followed by vomiting and loss of weight, a mild disturbance in the gastrointestinal tract having continued during the time the otitic infection was latent.

Second, after the diarrhea was established at eight months of age, the increasing severity of the attacks with continued dietary and general treatment, and the rapid recovery following elimination of mastoid infection.

Third, the distinctive type of X-ray findings, namely, a mastoid space containing air and evidence of increased density in the surrounding bone.

This case represents the unusual from the standpoint of rapid and uneventful recovery. In most of our cases the wound has not healed under three weeks, and the period of convalescence has averaged ten weeks. The bacteriologic findings have been interesting. In twelve cases pure cultures of hemolyticus staphylococcus aureus have been found; in sixteen, streptococci of the hemolytic type were found, two only of which were in pure culture.

#### IMPRESSIONS.

1. That mastoiditis may occur coincidental with an acute respiratory and gastrointestinal infection and may become an important factor in the outcome of the case; that in others, the mastoid may become involved and remain as a focus of infection producing gastrointestinal symptoms of a toxic nature or subsequent intestinal infection.

2. That surgical intervention cannot be decided upon intelligently without close cooperation between the pediatrician, the otolaryngologist and the roentgenologist, but that no mastoid should be opened without clinical evidence of disease in the mastoid space.

3. That adequate preoperative and postoperative treatment is of vital importance.

4. That all infants with marked or continued gastrointestinal disturbance should have careful ear examinations, and if pathology is found, early and free drum incision should be made. If the mastoid is involved and satisfactory improvement is not had by ear drainage a mastoid operation is indicated.

5. That operation, except in the occasional case, should not be attempted during the violent gastrointestinal crisis.

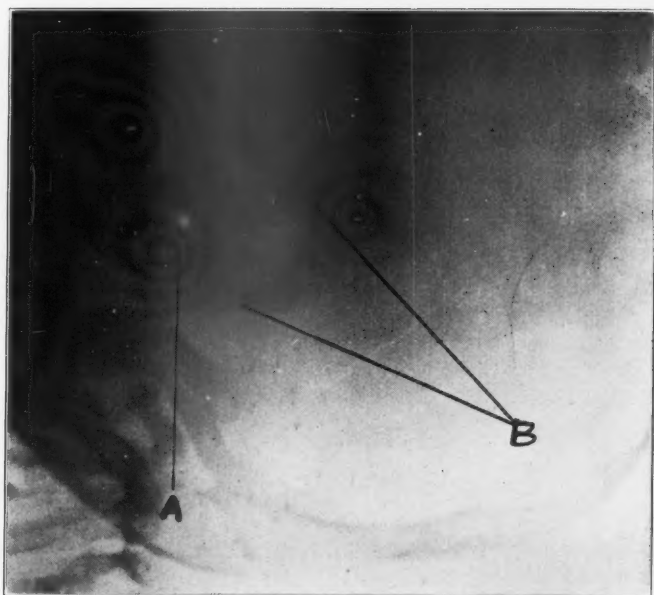


Fig. 1. Roentgenogram of normal mastoid in child 7½ months of age.  
A, auditory canal; B, outline of pneumatic mastoid space.

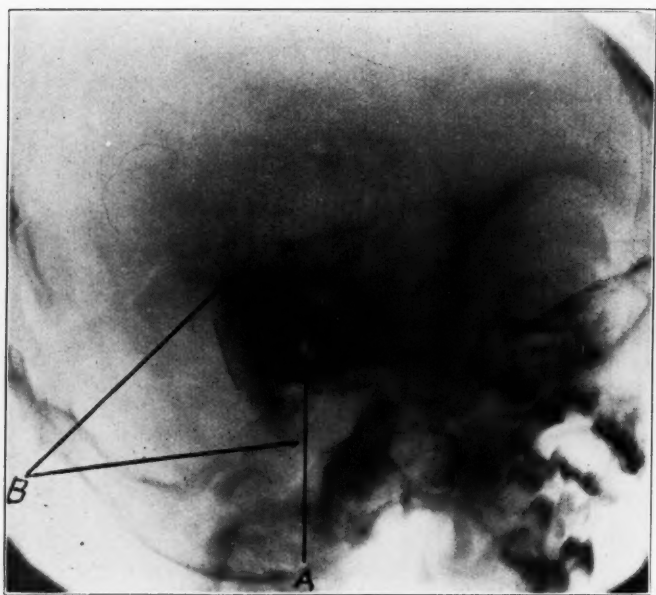


Fig. 2. Roentgenogram of 7 months mastoid from infant with violent dehydrating enteritis. This cavity contained swollen edematous mucosa and purulent material. Infecting organism, hemolytic streptococci. A, auditory canal; B, outline of mastoid space showing generalized increased density.

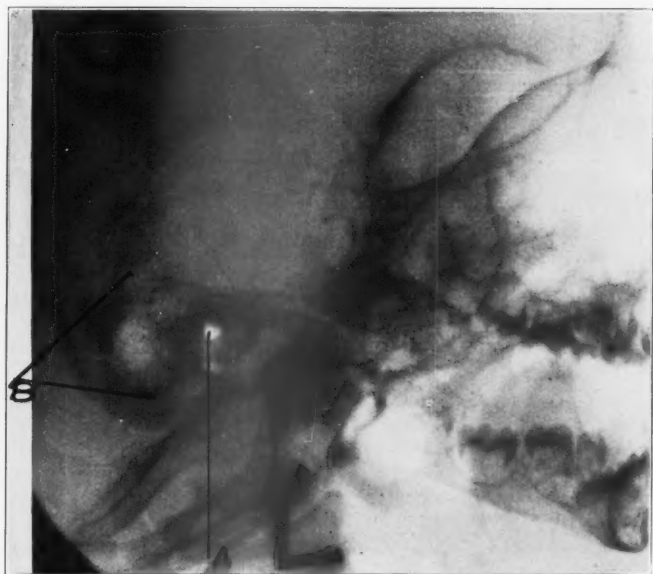


Fig. 3. Roentgenogram of mastoid space in case reported, 8 months of age. This cavity contained air and was lined with a moist gray necrotic bony ball. The mucosa was destroyed. Infecting organism, hemolytic staphylococcus aureus. A, outline of mastoid space, showing unusual transparency through the cavity with increased bone density surrounding.

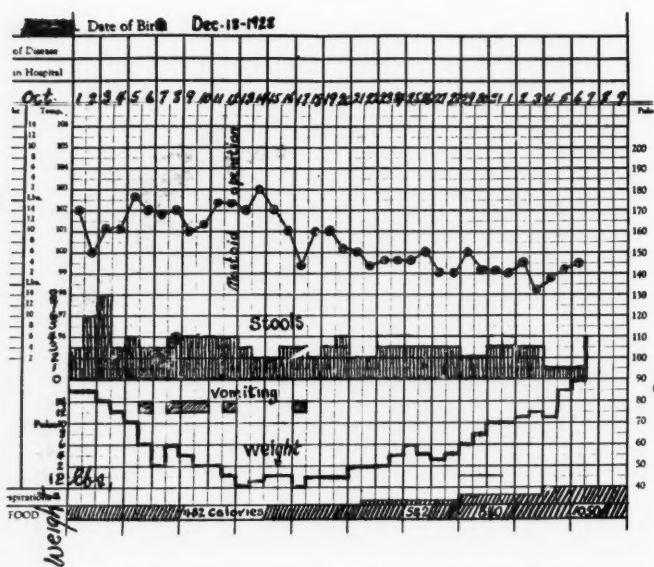


Fig. 4. Chart of case reported. Infection in both ears at two weeks. Continued gastrointestinal disturbance, vomiting, etc., until eight months of age, when diarrhea began. Rapid complete recovery immediately after mastoid spaces were cleaned out.



V.

SUDDEN ALLERGIC REACTIONS LOCALIZED IN  
THE ANTRUM.

BY ARTHUR W. PROETZ, M. D.,\*

ST. LOUIS.

The lining membrane of the maxillary sinus can, under the influence of an allergic reaction, increase in thickness from a fraction of a millimeter to as much as one centimeter overnight.

The appearance of polyps, of diffuse edema, and of similar filling defects commonly noted in radiographs of the antrum, has long been regarded as the result of chronic inflammation, and in many quarters is considered sufficient evidence of hyperplasia to justify more or less radical operative interference.

Examination of sinuses injected with certain radiopaques discloses the fact that a membrane may swell to several times its normal thickness between the time of the original injection and that of the seventy-two hour plate. The percentage of individuals reacting in this fashion varies from 5 to 20 per cent approximately, depending upon the radiopaque employed.

This observation is based upon a series of radiographs examined over a period of four years. During the first three years of this period a single radiopaque was employed, which was diluted one-half with olive oil. From this series, only an occasional instance of rapid thickening was encountered. One year ago a new radiopaque, then first appearing on the market, became the medium of choice. This was undiluted, but is a halogen compound of certain esters of the acids of olive oil. In the new series about one in five patients shows a demonstrable thickening of the membrane between the original exposure and the seventy-two hour plate. This was at first thought to be due to the possible irritating action of bromin on the sinus membrane, but in the light of the three following cases I now believe it to be allergic.

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\*From the Department of Otolaryngology, Washington University.

The first case is that of a woman of about forty years, originally seen by the allergist because of recurrent attacks of asthma. Not responding to any skin tests, the patient was referred for nasal examination. Beyond a moderate pallor and a few eosinophiles in the secretions, the nose showed nothing; air passages were free and the usually abundant mucus was absent. X-ray examination was negative. For some reason which I am unable to determine, the right antrum was filled with equal parts of lipiodol and olive oil through a trocar. Bone shadow and oil shadow corresponded perfectly and the sinus was pronounced negative. At this time a filling by displacement was done, using the same radiopaque. The posterior sinuses and the opposite antrum were also negative. (Figs. 1 and 3.)

During the seventy-two hour period awaiting the second plate, the patient was permitted to leave the hospital. She visited a relative, slept on a goose-feather pillow and suffered a severe attack of asthma without nasal symptoms. Films made the following morning (Figs. 2 and 4) showed a membrane approximately a centimeter thick, which had extruded most of the oil, and presented the typical appearance of a polypoid antrum. The other sinuses remained unaffected.

That this was not a reaction of the oil itself may be concluded from the fact that there had been some oil in the affected antrum for five days without change and that the thickening appeared only after the asthmatic attack.

I am unable to explain the reason for the reaction in this single cell which did not take place in the others. It is believed by allergists that local trauma may set off a reaction, and the trocar puncture at first resorted to in this case may have been responsible here. The fortunate coincidence of the asthmatic attack occurring during a progressive daily radiologic examination of the patient strengthens the likelihood that the reaction was purely allergic. Following is the allergist's record of the case:

Mrs. C. M. F., seen September 28, 1929. History of winter colds since 1918, which consist of mild nasal blocking, nasal discharge, usually colorless, and sneezing. No itching. Summers: always been well until this summer, when nasal symptoms persisted throughout the summer. Patient is allergic individual, in that ingestion of quinin is followed by urticaria. Mother and two maternal cousins also have idio-

syncrasy to quinin, as has the patient's oldest child. Four paternal uncles have bronchial asthma. She always has had asthma, in addition to her nasal symptoms, at or immediately following Thanksgiving, Christmas and New Year's and during the month of March.

Since the development of symptoms following deliberate exposure to feathers, it is probable that the reason for the symptoms at these times is due to her dressing the fowl which is eaten at these festivals. The symptoms during March can be accounted for by the necessity of cleaning the chicken houses at that time. Also, this is the first year that they have kept ducks, which must be plucked every six weeks, thus accounting for the symptoms occurring during the summer time for the first time this year.

November 19, 1929.—Occasional mild symptoms. Food diary discloses onions or chocolate as the probable cause. Has had no incapacitating bronchospasm or nasal symptoms since the experimental observation noted above.

December 9, 1929.—Two deliberate exposures to feathers followed by nasal and chest symptoms. Also, suspect wheat, whole Irish potato and overdose of milk.

December 20, 1929.—Nasal symptoms following exposure to wool (sewing on wool coat). Clinically better since last note which is coincidental with complete wheat avoidance.

January 11, 1930.—Ate wheat (light bread) December 27, 1929; no worse.

February 28, 1930.—No further symptoms. Patient cannot be induced to return for experimental repetition of sinus filling and exposure to goose feathers. (C. H. Eyermann, M. D.)

A second case has come under observation which is probably similar to the first. In this case an extreme thickening of the maxillary mucosa occurred between the first sinus filling with brominol by displacement and the seventy-two hour period. (Figs. 5, 6, 7, 8.) The reactions were not so sharply defined as to be able to attribute the sudden thickening definitely to allergy except that they occurred in an allergic individual; definite exposure to the specific allergens could not be elicited in the history.

There was seen recently in consultation, a patient whose left maxillary antrum on radiographic examination with injected lipiodol was obviously filled with polypoid material. The cavity was so small that scarcely any lipiodol could be introduced through a cannula. These findings, added to a very complete history of pain along the second division of the fifth nerve, prompted a radical operation on this antrum on the day following the X-ray examination. To the operator's surprise, the lining membrane was now quite thin, with the exception of an insig-

nificant, spongy area in the vault of the cell. A subsequent examination for allergy disclosed the fact that the patient was susceptible to chocolate, beef, lamb, pork, barley, pepper and milk, horse dander, orris root, dog, cattle and feathers by the intradermal method. By the cut method, definite positive reactions were obtained to the pollen of black walnut and to dog epithelium.

#### SUMMARY.

1. The antral mucosa, in allergic individuals, may increase to ten or more times its normal thickness during and allergic attack.
2. The thickening may be confined to a single sinus.
3. Allergic edema of the antral mucosa can probably subside within a few hours, an hypothesis justified but not proved by case three.
4. Obliteration of the maxillary sinus, as demonstrated, by X-ray findings, is not necessarily hyperplasia, and allergy should be excluded before operation is undertaken.
5. A mineral oil is the logical diluent for radiopaques to be used in allergic individuals.

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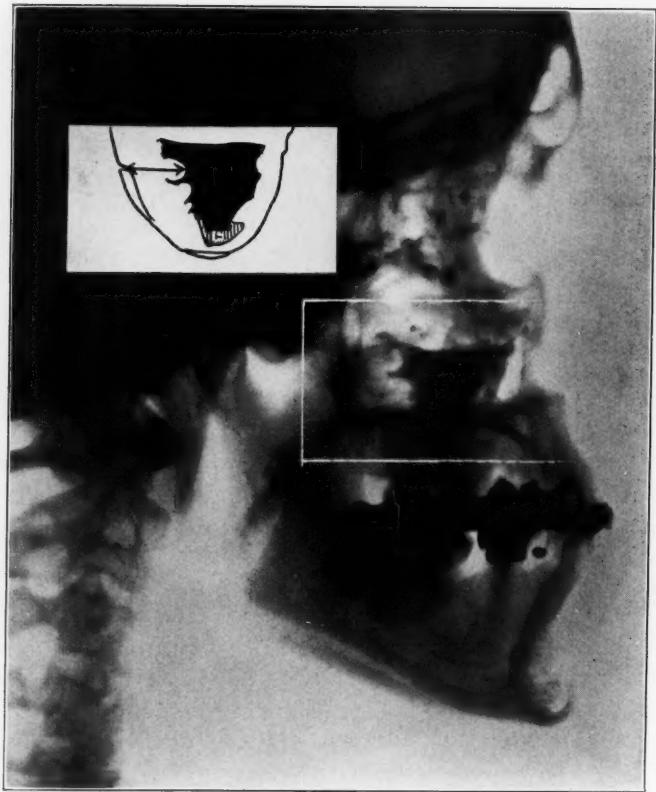


Fig. 2. Case 1. Lateral film made seventy-two hours after Fig. 1 and twelve hours after acute asthmatic attack. Note the extreme thickening of the mucosa indicated by the the arrow in the diagram.

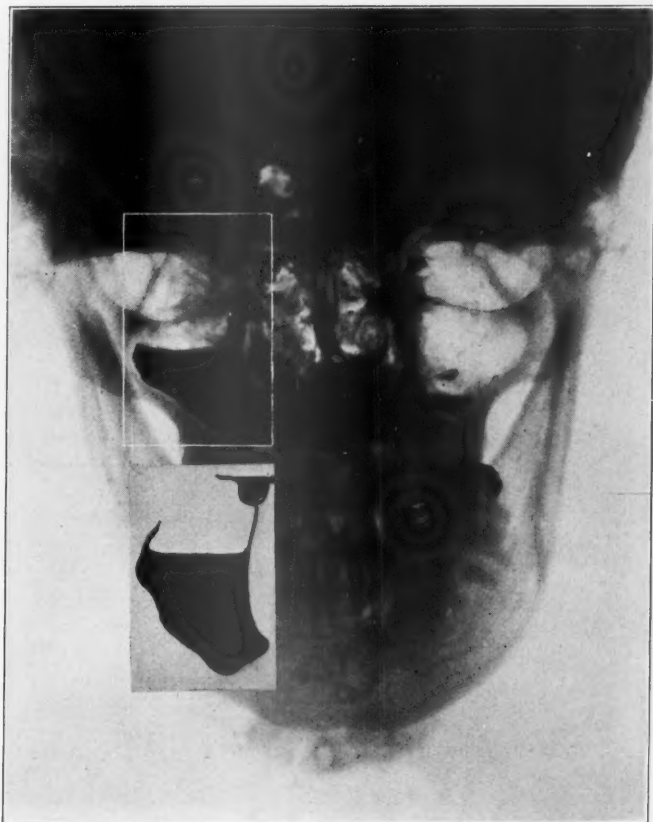


Fig. 3. Case 1. Posteroanterior view of Fig. 1. Note again the thin membrane in the antrum and also in sphenoid and ethmoid cells above.

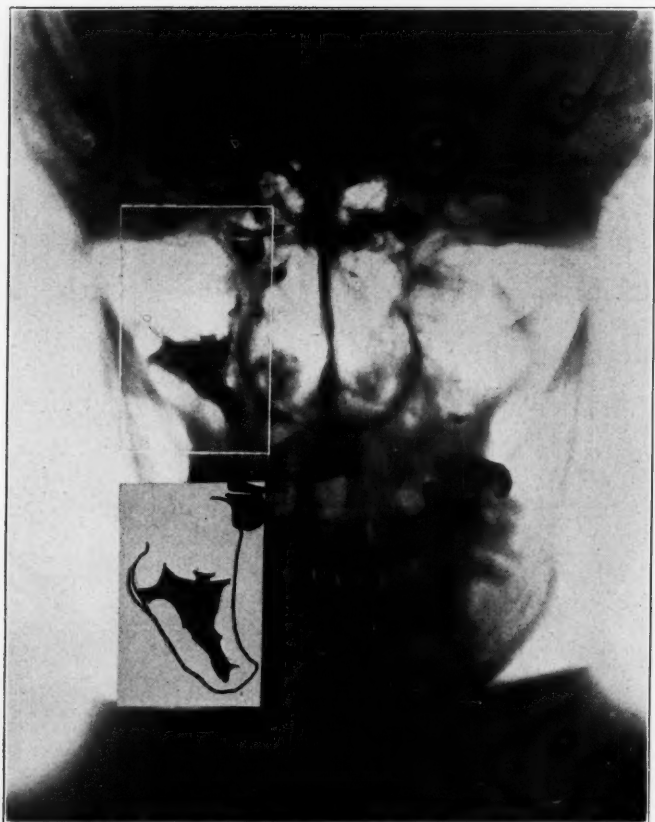


Fig. 4. Case 1. Postero-anterior view of Fig. 2. Note extreme thickening of antral mucosa while that of ethmoid and sphenoid remains unchanged.



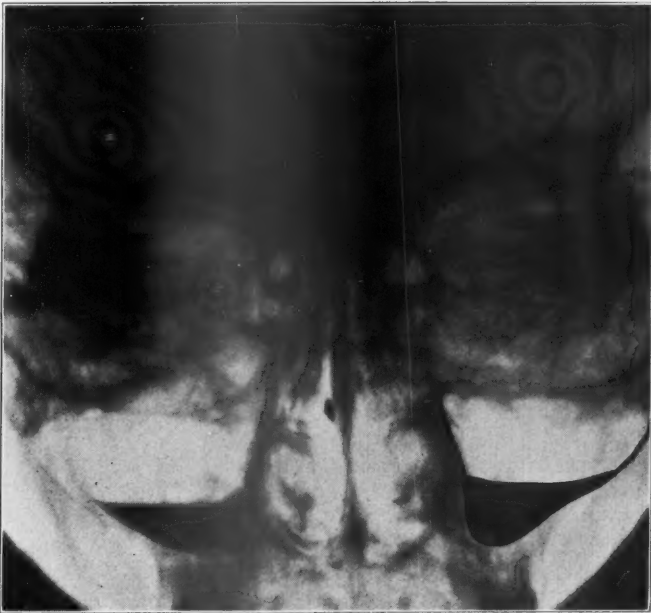


Fig. 5. Case 2. Initial film. Right antrum is unretouched. The left is diagrammatically represented.



Fig. 6. Case 2. Seventy-two hour film of the preceding.

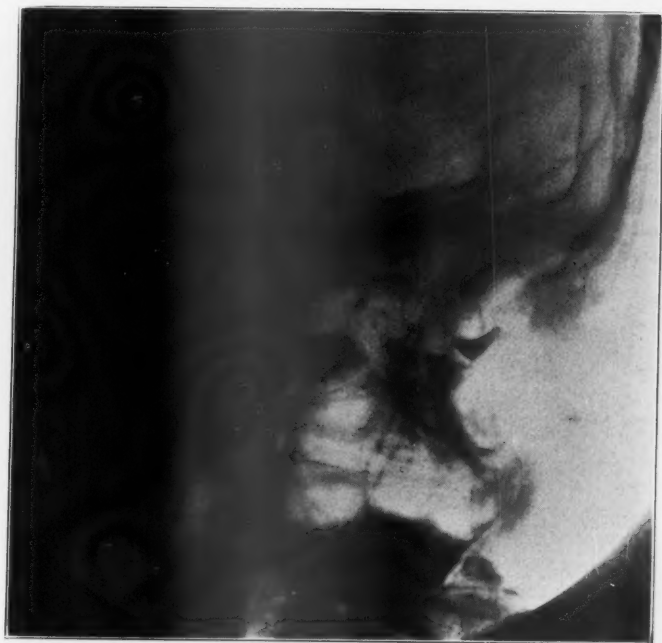


Fig. 7. Case 2. Lateral view of Fig. 5.



Fig. 8. Case 2'. Lateral view of Fig. 6.

## VI.

### MACROGLOSSIA WITH AN ASSOCIATED MICROGNATHIA CAUSING RESPIRATORY DIFFICULTY.

BY H. M. JANSE, M. D.,\*

ST. LOUIS.

This case is reported for its interesting clinical aspect and because of the simple, rather unique method of handling the patient as worked out by the nurse in charge.

C. K., age eight weeks, was a full term, normally delivered infant. She had a cleft palate, was unable to nurse at the breast, and was immediately put on artificial feedings which were taken with considerable difficulty.

The infant had attacks of dyspnea and cyanosis soon after birth. On each occasion the nurses were able to give relief by picking the infant up by her feet and allowing the head to hang. The first time the mother saw the infant she was struck by the fact that the infant breathed hard and that there was a sinking in of the chest with each inspiration. These attacks gradually became more severe and frequent. They often were precipitated by the taking of food. The day previous to admission to the hospital the patient had five very severe attacks.

The physician in charge of the case had made a diagnosis of enlarged thymus.

The patient was admitted to the St. Louis Children's Hospital November 24, 1929. The important things noted by the pediatricians on examination were: (1) The infant was extremely malnourished, (2) there was an inspiratory stridor, (3) receding type of chin, (4) wide cleft in hard palate, (5) marked retraction of sternum with each inspiration.

X-rays did not show an enlarged thymus. Blood Wassermann was negative. Nose and throat cultures were negative.

Patient was referred to nose and throat department for laryngoscopic examination to see if the cause of the respiratory difficulty could be determined.

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Direct laryngoscopy was twice attempted, but due to the fact that the tongue was enlarged and in close proximity with the posterior pharyngeal wall, it was impossible to see the larynx. On each occasion the infant stopped breathing, and it was only with considerable difficulty that the patient was revived. Following the second attempt it was necessary to administer artificial respiration, carbon dioxide and oxygen. It was only after the tongue was grasped and retracted that the patient quickly recovered. It was now noted that on retracting the tongue the breathing became normal and the sternal retraction disappeared, but upon releasing the tongue it would fall back, blocking the oropharynx, causing the difficulties to reappear.

The orosurgical service was now called in consultation. They placed three catgut sutures through the anterior portion of the tongue, and gave instructions for the mother to remain with the child, putting light traction on these sutures. It was at this time that the nurse constructed an apparatus which she felt would better care for the infant and relieve the mother of her duty. She increased the length of the sutures, passed them through a suspended pulley and tied a small specimen bottle on the end. In case traction needed to be increased, water was put into the bottle or released (Fig. 1) by pouring some of it out.

When the infant was seen in the afternoon she seemed contented, was breathing easy, had been sleeping, and now took her feedings quite normally.

A note was made by the pediatrician, dated November 26, 1929, to the effect that the baby slept most of the night, was breathing normally this morning.

November 27th the baby continued to breathe all right, but with release of traction the tongue still tends to close off the oropharynx and cause obstruction to breathing.

On November 28th the sutures started pulling through the tongue. This necessitated a tracheotomy.

Later the infant developed a bronchopneumonia and died December 2d.

The autopsy was performed by Dr. E. Burns of the Pathology Department. His findings are as follows: (1) The tongue was much larger and thicker than normal, this being

(Fig. 2) especially true at the base; (2) The thymus was of normal size and weight; (3) There was a bronchopneumonia. Microscopic sections of the tongue revealed a simple muscular hypertrophy without any cellular infiltration.

Butlin<sup>1</sup> divides the cases of macroglossia into three groups: (1) lymphangiomatous, which is most common; (2) simple muscular hypertrophy; (3) secondary hypertrophy of inflammatory origin. The muscular hypertrophy may be partial with symmetrical enlargement, or even unilateral with hyperplasia of one-half the tongue. There are cases where the muscular and lymphangiomatous types occur in combination. Microscopically the muscular types usually show either an increase in the number of fibers or an increase in the size of the fibers. These cases tend to remain stationary unless influenced by secondary inflammation.

Robin<sup>2</sup> reports a condition where obstruction of the oropharynx may be due to a backward lowering of the root of the tongue associated with the receding type of chin.

In this case there were probably two factors which entered in to cause the obstruction to breathing: first, the macroglossia, and second, the receding type of mandible. This resulted in the tongue being displaced posteriorly and its root lowered.

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Fig. 1. Infant with tongue suspended.



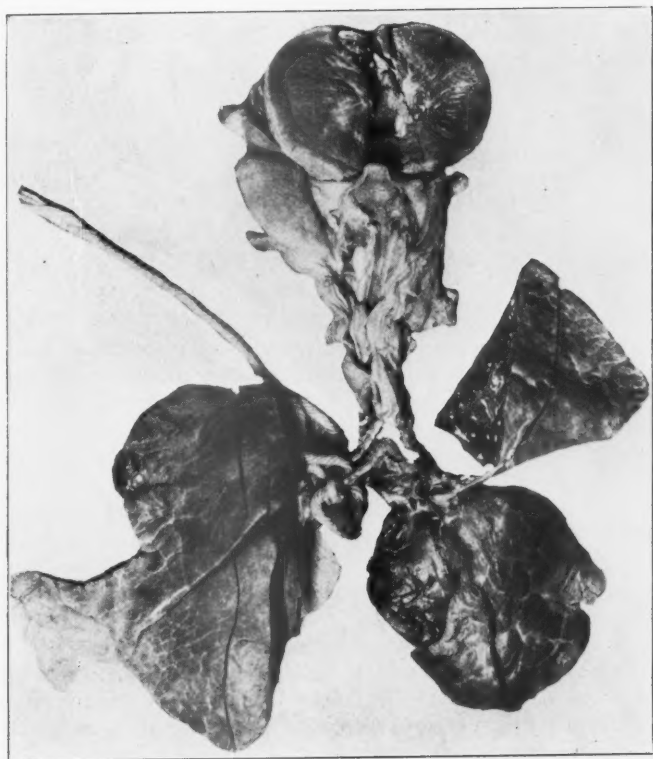


Fig. 2. Tongue split longitudinally, showing the hypertrophy.

## VII.

### THE LABYRINTH: A DISCUSSION IN A GENERAL WAY.

BY CL. F. WERNER, M. D.,\*

HAMBURG.

It is not the purpose of this paper to go very deeply into the subject of labyrinthine structure and function. It is purely a discussion in a general way, from our researches, of some of the most perplexing labyrinthine problems. In the last few years new facts concerning the labyrinth have been brought forth, which have further shaken the already weak foundations upon which our theories of labyrinthine function have rested.

The scientific study of the labyrinth is an especially difficult one. It is at once apparent that we cannot experiment on humans directly. We must wait for our material in the autopsy room. Many autopsies are unobtainable, and often when consent is granted hours and often days pass before the autopsy is performed and the temporal bones obtained. By this time postmortal tissue changes have destroyed in large measure the delicate tissues we wish to examine.

Thus we can come much closer to our goal when we take the longer way of animal experimentation. For this purpose mammals are the best suited, in as far as the routes for aural infection are the same as in humans. That is, they have not only an inner ear but a middle and outer ear as well. However, since pathology is based on a knowledge of the normal structure and function of an organ even experimentation on mammals is in many ways poor because:

1. The membranous labyrinth is relatively small and embedded in very compact bone, making operative experimentation extremely difficult;
2. Many fine histologic details are poorly recognizable.

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I wish to thank Dr. Conrad Gale for his help in translating and helping to prepare this paper.

It is therefore best to experiment on the lower vertebrates, and especially fish. The labyrinthine study in fish opens up an entirely new perspective and answers many questions which through mammalian study remained in doubt. The reason for this lies in the fact that the labyrinth in fish is extremely large, easily reached, and very accessible for experimental operations. Fish have no outer or middle ear, and the labyrinth is of primary importance and highly developed. In fact, so highly developed that by comparison the human labyrinth is primitive. This may sound absurd, since we are accustomed to think of man as the most highly developed organism. But it is true, and therefore it will be very interesting to note some of the most noteworthy points of the piscine labyrinth.

There are fish whose brain is not much larger than the labyrinth, and one type of shark has been known to have a labyrinth about 14 cm. long (Kolmer and Eisinger, 1925). This results from the fact that the labyrinth continues to grow throughout the life of the fish. This point has been definitely proven by measurements which we have established in 1926. In contradistinction to this a newborn child has a labyrinth fully as large as an adult (Tremble, 1929). The deduction to be drawn here is that in humans the labyrinth has lost its postembryonal growth, and its relative size thereby has been much reduced (Fig. 1).

This reduction in size of the human labyrinth is not the only result, since with it has come a simplification in its structure. The human membranous utriculus is of comparatively simple structure, from which lead the three semicircular canals (Fig. 2). It is also connected with the sacculus. This membranous structure, in some fish, as, for example, the shark and ray, is very much more complicated because of additional folds and relationships. Retzius has already shown that the utriculus of these fish is divided into two distinct parts, often separated widely from each other (Fig. 2).

In our study of the labyrinths of about thirty species of sharks and rays we have found many interesting variations which, because of the nature of this paper, cannot be gone into. In fish which possess an especially shaped head, as, for example, the hammerhead shark, we find an unusual shaped labyrinth. Since the labyrinths of these fish are not embedded in

compact bone we can see where conditions, as the shape of the head, etc., could have influenced them. In contradistinction to this the vestibular part of the labyrinth in man and mammals has a simplified, uniform construction. The noteworthy fact to be drawn here, and which by otologists is especially overlooked, is that what we deduct from the human labyrinth cannot be held as sound for all labyrinths. In fact, we must first really know the labyrinths of all vertebrates, and then only can we come to know the universal laws of labyrinthine physiology. Once possessed of these universal laws we can really understand their many variations.

Especially highly developed in fish is the otolith apparatus. In bony fish the otoliths are not many and small but consist of one large otolith. Since the otolith grows yearly in concentric layers the age of the fish can be told from its otolith. Studies which we have made in 1927 and 1928 show that the otolith apparatus is very highly differentiated. On every macula both an anterior and a posterior part can be seen. These two regions are entirely different. The anterior has a high epithelium and is not covered with the otolith, the posterior vice versa. Often both parts are innervated by separate nerves. Most highly developed is the macula utriculi. It is only in the last few years that these differences have been found in mammals; only here the differences are not very definite and extremely difficult to discover. Surely had the otolith apparatus in fish been studied earlier, then the discovery in the mammal labyrinth would also have been made earlier.

These varied anatomic studies have brought forth many theories as to function. This as yet is far from being settled, though textbooks quote them in a positive manner. For example, there are many discussions as to whether the otolith produces its stimulus by pressing on the sensory hairs, pulling on them, or by bending them to and fro. The school of Magnus and de Kleijn, in its belief that the stimulus is produced by a pulling on the hair cells, is most numerous.

However, we are not of their opinion, because we believe that the uncovered part of the macula cannot receive its stimulus in this way. Our theory of hydrostatic pressure causing the stimulus meets all the above objections and has been already in part proven to our satisfaction by experimentation.

In this way, both parts of the macula receive opposite stimuli, one positive, the other negative.

When, in mammals, the membranous labyrinth is opened and the hydrostatic pressure of the endolymph lessened, we found, after Wittmaack (1929), that the sensory end organs, namely, the macula and crista, swell. The above also occurs in fish when the otolith is removed from the macula (Fig. 3). In some fishes, as, for example, the carp and the herring, the inner ear is directly connected with the air bladder. In this way, as the fish swims deeper into the water, the increased pressure of the water increases the pressure in the air bladder, which is transmitted hydrostatically to the macula of the inner ear.

We believe, therefore, that in all vertebrates and in man hydrostatic pressure is the physiologic stimulus for the macula.

Equally erroneous have been the theories until now held of the relationship of the macula sacculi and macula utriculi to one another. In man and the higher vertebrates the two maculae have nearly the same histologic construction; only their relation is perpendicular to one another. It was deduced that both had the same functions, only exercised in different planes.

Just exactly the part played by the macula sacculi or macula utriculi has never been known, because of the extreme technical difficulties involved in operating on the mammalian labyrinth. It is only some years ago (Versteegh, 1927) that the macula sacculi has been definitely destroyed in the rabbit without injury to the macula utriculi (shown later on microscopic sections). Destruction of the macula sacculi in no way influenced the normal labyrinthine function. No reflex was changed or destroyed. These results are especially astonishing in view of the extremely thorough and scientific work of Magnus and deKleijn.

However, for years the fact has been known that in fish one could remove the sacculus otolith without in any way injuring labyrinthine function. Very recently the fish experiments of Maxwell (1923) proved definitely that all the reflexes of position proceed from the macula utriculi. Also our experiments with small salt water fish (1929) show that removal of the sacculus otolith after half an hour has no effect or reaction.

Here also the otolith is so large that by comparison the human otoliths in bulk would measure the size of a golf ball.

Until now many researches on mammals have misled us. Perhaps this might have been avoided if we had observed more closely the lower vertebrates. Most important is the fact that theories based on limited material and scope do not necessarily have universal application.

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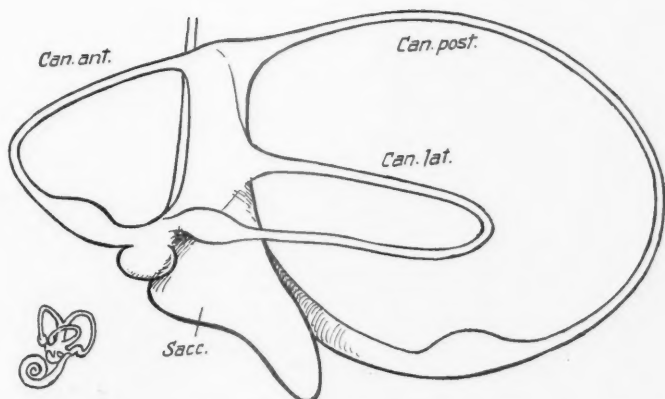


Fig. 1. Comparative size of human and piscine (Greenland shark) labyrinths, lateral view. Can. ant., lat., post., canalis semicircularis anterior, lateralis, posterior. Sacc., sacculus.

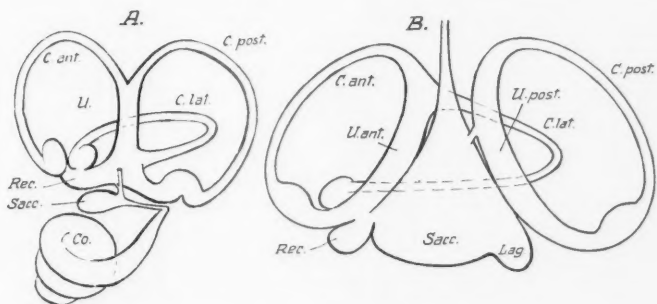


Fig. 2. Schematic: A., mammalian labyrinth; B., shark labyrinth, C. ant., lat., post., canalis semicircularis anterior, lateralis, posterior. U., utricle; U. ant., Utriculus anterior; U. post., Utriculus posterior. Rec., Recessus utriculi; Sacc., Sacculus; Lag., Lagena; Co., Cochlea.

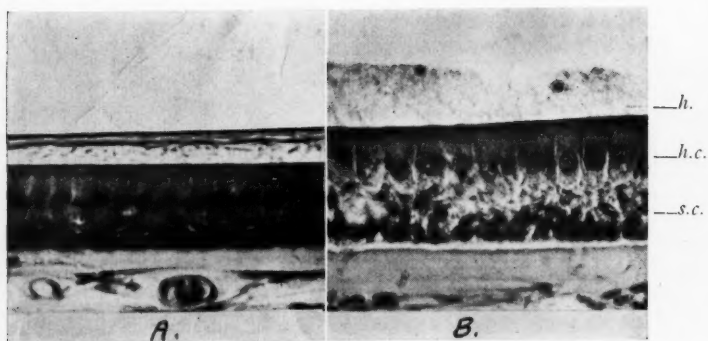


Fig. 3. Macula sacculi (piscine). A, normal; B, macula showing distinct cell swelling after otolith removal. h., sensorial hairs; h.c., hair cells; s.c., supporting cells.



## VIII.

### SCISSORS POINT EMBEDDED IN THE INNER TYMPANIC WALL, WITH FACIAL PARALYSIS.

BY V. V. WOOD, M. D.,

ST. LOUIS.

Case report: Patsy H., aged four years, first seen November 21, 1925. In February, 1925, a window fell on the child's head. Immediately a seventh nerve paralysis was noticed on the right side. A suppurative discharge from the right ear appeared a few days later. The seventh nerve paralysis and aural discharge had persisted without improvement.

Dr. V. P. Blair referred the patient because of the ear supuration which he wished to have cleared up before attempting any kind of facial anastomosis. It was the opinion of all, including a neurologist, that the nerve would not undergo spontaneous repair after such a long period of time (about nine months) without any apparent improvement.

Aural examination, November 21, 1925: Very profuse purulent discharge of a foul odor from the right ear. When the ear was carefully wiped out it was found that the posterior quadrants of the right drum were missing. The discharge was pumping through this large perforation in considerable quantity. There was a complete paralysis of the facial nerve on the right side. The patient was unable to lower the right upper eyelid or wrinkle the right forehead, and all muscles supplied by the seventh nerve on this side were immobile. The child was intractable and no cooperation for accurate hearing tests could be obtained. However, it was apparent that she was probably stone deaf in the right ear.

Douching the right ear produced no nystagmus or evidence of vertigo. It seemed evident that the labyrinth on the right side was dead or its connection with the central nervous system was severed.

It was the opinion of the writer that when the window fell upon the child's head a basal fracture had resulted and that one line of the fracture had gone through the internal audi-

tory meatus and caught the seventh and eighth nerves together. Because of the character of the aural discharge and the complete absence of hearing, together with absence of vestibular reactions, a radical mastoid was recommended to clear up the suppurative process prior to any attempt at a facial anastomosis.

Prior to the radical mastoid operation an X-ray picture was ordered as a routine procedure. The pictures showed a small angular opaque foreign body, the situation, size and shape of which are illustrated in the accompanying reproduction of the X-ray picture.

Much conjecture resulted as to the character and manner of entry of this foreign body. The writer felt that probably a piece of leaded window glass had entered the ear when the window fell and its glass was cracked.

A radical mastoid operation was done, and a small piece of metal was found embedded in the internal tympanic wall in such position as to sever the facial nerve as it ran downward in its third direction through the fallopian canal. In spite of the suppuration about it, the foreign body was still so firmly embedded in the bone that considerable traction was necessary to dislodge it after being grasped with a strong forceps. The piece of metal was a scissors point. It showed the beveled sharp cutting edge, one side rough, dull and slightly rusted, and the other three sides not rusted, but shiny and evidently well plated.

The family then remembered that the scissors had been found with one point broken off several months before. It was easy to fit the tip to the broken blade. They said that the scissors were often left, points upward, in a crack in the window sill. It is evident that when the window fell one of the upturned blades of the scissors entered the external auditory canal, pierced the drum and inner tympanic wall, severed the seventh nerve in the fallopian canal and was then broken off and left embedded in the bone. In the excitement of extricating the child from the window the presence of the scissors escaped notice.

This case is reported for three reasons. First, to exemplify the wisdom of always having a roentgenogram taken prior to operation in either a simple or radical mastoidectomy. Even

though an X-ray picture may be days behind the development of the pathologic process in an acute mastoiditis and thus lead one to underestimate the urgency of operation, or vice versa, and even though it may be chiefly of value in showing unusual anatomic conditions and location of the sinus, in chronic otitis media and mastoiditis it is, nevertheless, a good rule always to order one taken because it may show some uncommon or unsuspected thing at any time.

Secondly, a facial paralysis due to severance of the nerve in its course through the fallopian canal, when the history clearly indicated a probable skull fracture, was a baffling coincidence if not an extremely interesting one.

Thirdly and chiefly, this case was reported because of its possibilities from a medicolegal standpoint. The potential medicolegal complications for the operator may be easily imagined. If a roentgenogram had not been taken as a routine procedure it is very doubtful if the foreign body would have been found and removed. It is surprisingly difficult to find a foreign body, even with an X-ray picture, resting before one's eyes. Only those who may have experienced such an exasperating difficulty will fully appreciate that statement. However, assume that the picture had not been taken and the scissors point had been missed at operation. It is certain that its presence would have caused the ear suppuration to continue. As a consequence the operation would have been a failure, and probably in the course of time the patient would have drifted into the hands of a more careful otologist who would have taken a picture, discovered the foreign body and removed it. The almost inevitable conclusion would have been that the writer had broken off and left the scissors point in the ear during the previous operation. As a result, it is probable that a malpractice suit would have been filed and that the operator would have been an innocent victim of circumstantial evidence.



Scissors point embedded in the inner tympanic wall.

## IX.

### A CONSIDERATION OF DISEASES OF THE BLOOD AND LYMPHATIC GLANDS IN RELATION TO OTOLARYNGOLOGY.

BY PERRY G. GOLDSMITH, C. B. E., M. D., C. M., AND  
GREGOR MCGREGOR, M. B.,

TORONTO, CANADA.

It is written that there abideth faith, hope and charity, but the greatest of these is charity, so Robert Hutchinson, an eminent British physician, in aptly applying this old but familiar text to medicine, makes diagnosis a matter of faith, prognosis a matter of hope, and treatment a matter of charity (too often so), but the greatest of these is diagnosis.

It is only by the intelligent interpretation of signs and symptoms that diagnosis reaches a firm foundation. It is all important if the treatment is to be intelligent and efficient. In fact, the first part of the treatment is diagnosis, the second is diagnosis, and the third is diagnosis.

In this interpretation of signs and symptoms the laryngologist has frequently to deal with bleeding from the nose and throat, and altered states of the glandular system shown in enlargements of the glands, such as those of the Waldeyer ring and the cervical neck chain. As such pathologic conditions rest primarily on alterations of the blood or blood-forming organs, we considered a study of case records showing the important and close relationship otolaryngology has to general medicine would be of interest and instruction. It must be remembered that the correct diagnosis of many obscure cases of hemorrhage from the mucosa of the nose and throat can only be made after skilled analysis of the completed blood picture. Often only the microscopic examination of a section of the gland affected can determine the underlying disease, but even skilled pathologists frequently have great difficulty in arriving at a diagnosis—for example, in cases of infective mononucleosis with ulceration of the tonsil.

In looking through the case records of the Toronto General Hospital, in those dealing with altered blood states in which otolaryngology has a part, it seemed simplest to divide them into several main groups: one in which bleeding was a prominent factor, another in which glandular enlargement seemed to dominate the picture, and still another in which neither of these signs was prominent and which we have taken the liberty to call the anemic group.

Group 1. (a) Congenital telangiectasis.

(b) Polycythemia vera.

(c) Myeloid leukemia (chronic and acute).

(d) Lymphatic leukemia (chronic and acute).

(e) Symptomatic purpura.

(f) Purpura hemorrhagica.

(g) Hemophilia.

(h) Salvarsan and benzol poisoning.

(a) In congenital telangiectasis there is a history of repeated and severe epistaxis for many years or even from childhood. Gradually weakness becomes prominent and only then does the patient seek medical advice. By this time anemia is very marked, the hemoglobin being as low as 20 per cent. The clotting and bleeding times are normal, so any necessary operation may be undertaken with safety. The diagnosis rests on the finding of reddish, spiderlike telangiectic spots in the mucous membranes and skin, which come in crops and fade on pressure. These spots are most commonly found on the septum in the region of Little's area and on the anterior portion of the turbinates. They are frequently on the tip of the tongue, and may be seen in the nasopharynx, larynx, and even on the tympanic membrane. Those on the septum bleed quite easily, and careful cautery or radium therapy does a great deal for the comfort of the patient and gives the blood forming organs a chance to recover.

\*(b) The florid cyanotic individual who presents himself with a history of repeated epistaxis should have a blood examination, for if he has a red blood count considerably above normal and an enlarged spleen, he is likely to be a case of polycythemia vera, provided there is no physiologic cause for the increase of red blood cells. The blood may even be so thick that it is difficult to get sufficient plasma for purposes of exam-

ination. Consequently death by thrombosis is not an uncommon termination.

(c) Not uncommonly in acute myeloid leukemia there is bleeding from the mouth or nose with ulcerations. The patient is very ill, with considerable elevation of temperature and marked splenic enlargement. The glands of the neck are usually enlarged. The white blood count may not be very high but will show a large percentage of very immature forms, such as myeloblasts, and a marked secondary anemia is present.

In the more chronic forms the spleen is also very large, but there is a very marked increase in the white blood count with a smaller percentage of immature forms. Bleeding from the mucous membranes comes in the terminal stage.

(d) Acute lymphatic leukemia resembles an acute infection with fever, swelling of the tonsils, ulcerative angina, and cutaneous hemorrhages. The glands of the neck enlarge and usually those of other groups, but the spleen is not very large. The blood picture with its high white blood count and large percentage of large lymphocytes makes the diagnosis. The acute types of leukemia are very similar, and it may be quite difficult to differentiate. However, differentiation is not important, for the prognosis in either case is rapidly fatal.

In the chronic forms of this disease small lymphocytes predominate the blood picture, there is a general glandular enlargement, and the patient is not very ill until the later stages.

(e) In symptomatic purpura it must be remembered that the ecchymoses and hemorrhages from the mucous membrane are only symptoms of some underlying condition, infectious, toxic, cachectic, severe anemic, neurotic or mechanical. The actual cause can only be determined by a careful and complete examination of the patient and the treatment must be instituted accordingly.

(f) Purpura hemorrhagica is a more severe form and may be rapidly fatal, with death from loss of blood or from hemorrhage into the brain. It differs from other purpuras in that the blood platelets are diminished in number. The bleeding time is much prolonged, the blood clot is soft and nonretractile, while in hemophilia bleeding time is not prolonged, the blood platelets are normal, and the clot retracts normally. The coagulation time in hemophilia is markedly prolonged, while in

purpura it is normal or nearly so. The application of a tourniquet to the upper arm results in the formation of petechiae on the forearm in purpura but not in hemophilia. Bleeding is most common from the nose and vagina.

(g) In hemophilia it is essential for a diagnosis that the individual should have been subject, more or less, to bleeding from various parts of the body throughout life. Hemophilia without demonstrable inheritance is rare, is not found in females, and is transmitted only by them.

(h) Agranulocytic angina. A toxic patient with sore throat, fever, chills, bleeding, sloughing ulcerations in the mouth and with a fetid odor to the breath may be mistaken for septicemia, but if examination of the blood shows the polymorphonuclear neutrophils to be almost completely wiped out, the case is likely to be agranulocytic angina. The lesions usually begin as small superficial ulcerations covered with a grayish or yellowish exudate which on removal leaves a bleeding surface. These may enlarge and increase in number very rapidly until they become large burrowing ulcers. The most common location is on the tonsils, which are usually inflamed and enlarged, and they may become so large as to make swallowing difficult. The pillars, uvula, soft palate, tongue, lips and even the gums may be involved. Even where the symptoms are not so marked one must be careful in giving a prognosis, for the outlook is almost hopeless.

One must be careful, however, that he is not dealing with another condition associated with an agranulocytic symptom complex, such as severe sepsis, aleukemia, leukemia, pernicious anemia, poisoning with benzol or salvarsan, and the effects of X-ray or radium therapy.

(i) Salvarsan poisoning may produce bleeding, sloughing ulcerations in the mouth, associated with a diminution of the polymorphonuclear neutrophils, but the history of treatment prepares the way to a diagnosis.

- Group 2. (a) Infections.  
(b) Malignancy.  
(c) Lymphatic leukemia.  
(d) Aleukemic leukemia.  
(e) Lymphosarcoma.  
(f) Hodgkin's disease.



- (g) Infectious mononucleosis.
- (h) Tuberculous adenitis.
- (i) Vincent's angina.
- (j) Actinomycosis.
- (k) Carotid body.
- (l) Fibroma.
- (m) Lipoma.
- (n) Tonsils.

(a) Local enlargement results most frequently from focal infections by way of the lymphatics, so that the area drained by the particular group enlarged must therefore be searched for the portal of entry. In an adult, in the absence of an obvious focus, the possibility of metastatic deposits must be considered.

(b) Seeing that malignancy of the mouth and esophagus is a frequent disease, the so-called sentinel gland in the neck in carcinoma of the stomach must be kept in mind. Glands in the lower cervical group on one side of the neck are frequently the first to be enlarged in Hodgkin's disease.

In generalized enlargement, where two or more groups are involved, the causative agent may be conveyed by the blood stream, as for example, in infectious mononucleosis, rubella, septicemia, secondary syphilis or bubonic plague. In this way the generalized enlargement has a definite diagnostic value.

If the glandular enlargement has arisen insidiously and if the patient's condition generally is not acute, the probable explanation lies in the direction of such diseases as leukemia, Hodgkin's disease, and rarely tuberculosis and sarcomatosis.

The specific features of glandular enlargement calling for special note are: The number and distribution within the group, the size, mobility and consistency, sensitivity, whether the glands are discrete or coalescing, and involvement of the skin or surrounding structures. When the glands are adherent to one another or to surrounding structures, it means the disease process has spread through the capsule, as it does in tuberculosis, pyogenic infection and malignancy. In chronic cases with several groups involved, the disease is likely to be chronic leukemia, Hodgkin's disease or rarely lymphosarcoma. Leukemia gives us the least difficulty in diagnosis because of a distinctive blood picture.

(c) The chronic lymphatic type of leukemia is characterized by generalized glandular enlargement, and a blood picture showing a high white cell count, 100,000 or more, in which the great majority are lymphocytes. The spleen may also be enlarged, although a large spleen and small glands are more characteristic of chronic myeloid leukemia, where the majority of the cells are of the myelocytic type. The individual nodes in a group are of much the same size, mobile, discrete, soft, and show no tendency to break down or involve the skin or other structures. They are insensitive to palpation and cause no discomfort apart from their size.

(d) The aleukemic type show a large glandular enlargement without a high white count, but with a relatively large percentage of lymphocytes, as in lymphatic leukemia. Tonsils are often enlarged both in the lymphatic and aleukemic types.

(e) In lymphosarcoma the tendency is for the glandular enlargements to be unequally transformed into large masses, the individual nodes showing marked variation in size. Their mobility is limited, and there is a tendency for the whole mass to be adherent to the deeper structures. Progressive enlargement of both tonsils is more likely to be lymphosarcoma than Hodgkin's disease. Pruritus, fever and sweating are absent, metastases are uncommon, and the spleen is not enlarged. Biopsy may be an aid in diagnosis.

(f) Hodgkin's disease. This often begins with enlargement of the glands in one side of the neck but it may begin in any group. The first symptom may be obstruction of the bowel due to enlargement of the lymphoid tissue in that location, or cough, or dyspnea from enlargement of the mediastinal glands. Swelling of the arm or abdomen may be due to pressure on the venous flow, or enlargement in the groin may simulate hernia. There is a tendency towards an orderly spread from one group to another. The glands are discrete, rather firm and elastic, not tender and do not involve the skin, while in metastatic carcinoma they are hard, irregular and fixed. Progress may be slow or rapid. The blood picture is not distinctive, though there is often an increase in the endothelial leucocytes. There is a great tendency towards infiltration of other organs. Fever, pruritus and sweating are common and the spleen is usually enlarged.

Biopsy may be an aid to diagnosis, but the interpretation of the microscopic picture is often difficult. The typical picture is an increase in fibrous tissue with a disturbance in the topography of the gland and the presence of multinucleated giant cells and eosinophiles. In these cases removal of tonsils is not going to cure glands in the neck.

(g) Infectious mononucleosis is a self-limited disease of young adults, usually males, characterized by an acute onset with fever, malaise, sore throat and glandular enlargement which usually begins in the neck. The tonsils are often inflamed and may be ulcerated. There is some increase in the white blood count, but the greatest change is in the percentage of lymphocytes, which is very high. This confuses the condition with lymphatic leukemia, but the acute onset with fever, sore throat and relatively low white blood cell count makes the diagnosis. In a few weeks the patient is back to normal and all anxiety is relieved. Biopsy is likely to be very misleading, as the picture is one of lymphosarcoma. As regards biopsy, Professor Duncan Graham, professor of medicine in the University of Toronto, makes the following statement:

"Among clinicians the impression is too widespread that the excision of a gland for microscopic examination is essential in the diagnosis of superficial glandular enlargement. This method for making a positive diagnosis has definite limitations. Too often the clinician advises the removal of a gland for microscopic examination without realizing that it is impossible for the pathologist to differentiate by histologic examination between glands from cases of lymphoid leukemia, aleukemia, lymphosarcoma or certain cases of Hodgkin's disease, without a report of the clinical examination of the patient and the results of the hematologic examination of the blood. This information alone is usually sufficient for the making of an accurate diagnosis. The microscopic examination of an excised gland is chiefly of value in the diagnosis of atypical cases of tuberculous adenitis."

(h) Tuberculous adenitis is most common in children and is associated with disease of the mucous membrane or enlargement of the tonsils. It is most common in districts where the milk is not pasteurized. The glands usually begin in the sub-maxillary group, and go on to form large knotted masses to

which the skin becomes adherent. Inflammation and suppuration may occur. Pruritus is absent and the spleen rarely becomes as large as it does in Hodgkin's disease. It is exceptional to find general tuberculous adenitis. If diagnosis is in doubt a gland may be removed for examination.

(i) Vincent's angina may produce marked enlargement of the cervical glands with tenderness, but the presence of deep ulceration, usually in the tonsil, from which the causative organisms can be found, will make the diagnosis. However, the ulceration due to malignancy must be kept in mind, for the Vincent's organism here may be a secondary invader. If there is any doubt it can be settled by biopsy.

(j) Actinomycosis may involve buccal, lingual or pharyngeal structures. It produces ulceration and marked infiltration with secondary enlargement of the cervical glands. Diagnosis is made bacteriologically, the causative organism being a streptothrix.

(k) At the level of the top of the thyroid cartilage occasionally there is enlargement of the gland known as the carotid body. These tumors are at first benign but may become malignant. The situation and the fact that it is a single enlargement give a clue, but diagnosis is made only by biopsy. It may be confused with (l) fibromata and (m) lipomata.

(n) A patient with a story of progressive enlargement of one tonsil or both is entitled to an examination which will exclude malignancy, though it may be quite innocent and due only to a chronic infective process.

#### Group 3. Anemic group.

- (a) Pernicious anemia.
- (b) Secondary anemia.
- (c) Chlorosis.
- (d) Hemolytic jaundice.
- (e) Banti's disease.

(a) A patient presented himself with the story that for several months previous he had been bleeding a great deal from the nose and that his eyesight was becoming much impaired. Careful examination established a diagnosis of pernicious anemia and his failing eyesight was due to old and new retinal hemorrhages. His left vocal cord was almost stationary, and there was a paresis of the internal tensor muscle. Careful ex-

amination by the medical department established a diagnosis of pernicious anemia, and proper treatment restored his voice and larynx to normal.

Other cases have various complaints, such as sore throat, sore tongue and headache. A blood and gastric analysis will establish a diagnosis and save a disappointment of local treatment to nose and throat.

(b) Removal of tonsils is not likely to restore bodily and mental vigor to the individual suffering from a marked secondary anemia, the cause of which may be in the rectum.

(c) Neither is it likely to cure the green sickness or chlorosis of young blondes who come from the factories of Europe.

(d) In Banti's disease the treatment of hemorrhages from the mucous membrane may be the removal of the spleen, for this condition is marked by bleeding from the mucous membrane, enlargement of the spleen, and a secondary anemia associated with a leucopenia.

(e) In long standing cases of hemolytic jaundice hemorrhage may be a prominent feature, but the primary factor in the disease is an increased fragility of the red blood corpuscles.

Not uncommonly cases with a hemorrhagic diathesis complain of deafness or vertigo. There is no reason why there cannot be a hemorrhage into the labyrinth as well as elsewhere, and this may be the cause of the symptoms. This can only be ascertained by labyrinthine tests and blood examination.

A few typical cases to illustrate may be of interest:

1. A middle aged woman, the mother of a medical student, was admitted to the hospital with a history of having lost a great deal of blood following the extraction of teeth. It was scarcely possible to see much of her face or anything in the mouth because of blood and the results of styptic medication. Careful examination revealed a very large spleen and a red blood count of over 8,000,000, a case of polycythemia vera.

2. A woman, aged 46, was cyanotic from obstruction to respiration, thought to be due to tuberculous inflammation of the larynx. Laryngeal examination revealed a normal larynx, but careful X-ray examination in the lateral position disclosed a very large mass in the mediastinum. This was believed to be Hodgkin's disease, and X-ray therapy reduced the mass and the respiratory distress disappeared.

3. A young man, aged 23, was sent to a sanatorium for investigation of the lungs because of fever, pallor and loss of weight. On arrival he developed pain and swelling in one cheek and involvement of the maxillary sinus was considered. The otolaryngologist was suspicious of a blood dyscrasia. He was able to palpate a spleen markedly enlarged, and blood examination revealed an acute myeloid leukemia.

4. Another man in a very important position was seeking relief from progressive weakness. He had also had repeated epistaxis. He had had his blood examined, and was told it was normal or even better than normal. The otolaryngologist who was consulted for the epistaxis noted the cyanotic appearance and asked for a complete blood examination. It was a case of polycythemia vera with a red blood count of 10,000,000. Venesection, X-ray treatment to the long bones and the administration of phenylhydrazin reduced the count.

5. A man presented himself for examination complaining of pain in the ear and swelling in the neck. He had large diseased tonsils, but at the base of the tongue there was a small ulcer which proved to be malignant and was the cause of his symptoms.

6. A medical student was ill with headache, fever, malaise and sore throat. His tonsils were large and inflamed, and there was a sloughing area in the upper pole of the right. A biopsy was done and the report was lymphosarcoma. However, in two months he felt as well as ever. It was a case of infectious mononucleosis.

In this paper no effort has been made to cover all the differential diagnostic points in these blood and lymph dyscrasias. It has been our endeavor to emphasize the importance of the medical examination necessary in otolaryngologic patients in whom our cross examination points to the possibility of a general systemic disease rather than a local disorder; but our consideration has not gone beyond the title of the paper. We have, therefore, made no reference to such conditions as hypertension, chronic hepatic or renal disease, uncompensated heart disease, infectious fevers and local lesions in the nose.

We are indebted to the Medical Service of the Toronto General Hospital for their cooperation and for the use of their records.

X.

MALIGNANT TUMOR OF THE BREAST WITH  
METASTASIS TO THE OPPOSITE SIDE OF  
THE LARYNX AND CONTRALATERAL  
VOCAL CORD PARALYSIS.

By W. V. MULLIN, M. D., AND

F. V. LANGSTON, M. D.,\*

CLEVELAND.

Metastatic carcinoma of the larynx is very rare, and, so far as I know, a case of metastasis from the breast has never been reported. Dr. A. L. Turner<sup>1</sup> reported a case of malignancy of the larynx in a male in which the primary tumor was a hypernephroma of the kidney. Epithelioma is the usual form of laryngeal carcinoma. On the other hand, medullary carcinoma is the most frequent type found in the breast. Consequently, the finding of a medullary carcinoma in the larynx in a patient who has a new growth of the mamma, is very significant.

The case which I am going to report had the additional interesting feature of having a vocal cord paralysis due to metastatic glands involving the left recurrent laryngeal nerve contralateral to the breast tumor.

The question arises as to how a contralateral metastasis is possible. The lymphatic pathway by which such a metastasis occurs has been carefully worked out by Dr. Turner.<sup>2</sup> The malignancy must travel from the breast to glands along the recurrent laryngeal nerve of the opposite side. Most of the mammary lymphatics empty into the axillary group of the same side. Some pass to the infraclavicular and some to the retrosternal glands. In turn, the axillary, infraclavicular and retrosternal glands drain into the supraclavicular group. The recurrent or peritracheal glands are closely associated with those in the supraclavicular fossa, and consequently they are

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\*Deceased.

very likely to become involved if the latter become malignant. This explains the homolateral vocal cord paralysis. Lymph vessels pass from the mammary glands across the midline to the supraclavicular and axillary glands of the opposite side, thus making it possible to have a contralateral metastasis.

About a dozen cases of vocal cord paralysis following malignancy of the breast, all of which have been in females, have been reported. In one-half of these cases the paralyzed cord was contralateral to the breast tumor. The primary reason for laryngologic consultation in each case was hoarseness, which developed on the average of three and one-half years following removal of a malignant breast. The paralyzes were due to involvement of glands near the recurrent laryngeal nerves.

Case Report.—The patient, a man, 58 years of age, first came into the clinic on September 11, 1928, at which time he complained of pain in his throat, difficulty in swallowing and weakness of his voice. These symptoms had been present for one month. One year before he had noticed a mass on the right nipple, which three months before he had injured, causing it to bleed. He had lost thirty-six pounds during the preceding six months.

On examination, the patient was found to be a well developed, somewhat obese man. His voice was weak and he vocalized with effort; he appeared weak and exhausted. There was a hard, circular mass about eight centimeters in diameter surrounding a small, punched out ulcer at the site of the right nipple. The supraclavicular glands on the right side were hard and slightly enlarged.

Laryngeal examination showed a soft bluish mass two centimeters in diameter extending upward from the left arytenoid region. The tumor did not seem to be intimately related to the surrounding structures, and there was nothing about its appearance that was characteristic of malignancy. The left vocal cord was immobile in the midline. The question arose as to whether the condition was paralysis of the left vocal cord due to pressure on the left recurrent laryngeal nerve, or to fixation of the left crico-arytenoid joint by infiltration of the laryngeal tumor.

A direct laryngeal examination was made. The tumor was freely movable, soft, showed no evidence of infiltration, and



could not have caused fixation of the left crico-arytenoid joint. No other part of the larynx was involved. A biopsy was performed. Pathologic examination proved the tumor to be medullary carcinoma, probably metastatic, from the right breast. X-ray plates of the chest showed a dense area in the lower mediastinum, probably metastatic. It was concluded, therefore, that the cord paralysis was due to pressure on the recurrent nerve by the mediastinal metastasis.

About one month later the patient became unable to eat. He declined rapidly and died October 29, 1928. A postmortem examination was not permitted.

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## XI.

### INTERMITTENT ABSENCE OF LEUCOCYTOSIS WITH ACUTE MASTOIDITIS: REPORT OF A CASE.

BY IRA FRANK, M. D., AND WILLIAM SMILEY, M. D.,\*

CHICAGO.

In textbooks, encyclopedic articles and monographs devoted to diseases of the blood and hematopoietic organs, the conditions described are mainly concerned with an increase of leucocytes or deviations in the number of red cells. Apparently there are no diseases in which a great depletion of the white cells is the prominent feature, or, perhaps they have not attracted attention. Usually the diminution of leucocytes is looked upon as an inconspicuous detail among other more important symptoms, although it has, as in influenza and typhoid fever, considerable importance in diagnosis.

In 1888 Ehrlich explained the leucopenia of a staphylococcal sepsis by failure of a protective leucopoiesis.<sup>1</sup> The changes in the blood characteristic of agranulocytosis were described by Türk about 20 years ago,<sup>2</sup> and, according to Philpitschenko,<sup>3</sup> severe damage to not only bone marrow but also other lymphoid tissues as a consequence of infections was given the name pannyelophthisis by E. Frank in 1915. The usual proliferative reaction was absent, so that anemia, leucopenia, disappearance of megacaryocytes from the marrow with amyelopoiesis, alymphopoiesis and activation of the cells of the reticuloendothelial system—phagocytosis of hemolysed red cells—all occurred together.

From such observations, it was but a short step for Werner Schultz,<sup>4</sup> in 1922, to launch the term agranulocytosis, applied to leucopenia, but also to a profound diminution or total absence from the circulating blood of leucocytes with granules in the cytoplasm. He suggested the term for these changes in

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the blood as they occur with sore throat, particularly the forms associated with gangrene, the Vincent's angina, the tonsillopharyngitis in which the spirillum of Vincent and the bacillus fusiformis are found associated. Schultz claimed this agranulocytic angina, angina agranulocytosa, is a disease entity. Many reports have been made in the few years since his announcement, and the profession at large is gradually becoming alert to the condition. A summary of the literature was made by Kaslin<sup>5</sup> in 1927. He reported two cases and reviewed the observations made by others in forty-three more. Recently Korach<sup>6</sup> has estimated the total number of reports as eighty, and still another review was that by Schultz<sup>7</sup> himself last year.

It is not our purpose to consider at length the many opinions advanced from time to time about this combination of alterations in the blood with local infections which frequently prove fatal. It is now accepted that in addition to gangrenous inflammations in and about the nasopharyngeal ring, similar forms of leucopenia take place with localized infections in many other parts of the body, especially the vulvovaginal region, anus, various parts of the gastrointestinal tract and externally in the skin. Although we have seen no accounts of studies of the leucocytes in noma or cancrum oris, it probably will be learned that this, too, is characterized by diminution of granulocytes.

There has been a definite drift away from the views which Schultz first advanced. In place of assuming that this symptom complex represents a distinct disease, agranulocytosis, there is a marked disposition among recent observers<sup>8</sup> to regard the diminution of particular forms of leucocytes in the circulating blood as an indication of a lowered resistance caused by changes already present in hematopoietic organs, and the severe course of the local inflammations—for the lesions are often multiple—as a result of unpreparedness to meet localized infections with the usual protective reactions.

A number of writers<sup>9</sup> have emphasized that preceding the high fever and other severe symptoms which frequently end in death after a few days, the patients are generally ill with malaise and loss of appetite; or when the localized disease is

angina, that there may have been a number of attacks of sore throat at intervals of a few weeks or months before the final outbreak of hemorrhagic and gangrenous inflammation. The mastoiditis we wish to report was accompanied by the usual leucocytosis. However, in place of a continuous high count of the white cells there were periods during the height of the infection when the count dropped to normal or close to normal. There was consequently no leucopenia in the strict sense of the term, certainly nothing approaching the conditions met with in agranulocytosis. But the difference, we believe, may be a matter of degree rather than one of character. If during infections that are regularly accompanied by leucocytosis there occurs a drop involving particularly the neutrophiles, the defensive mechanisms are generally considered inadequate. This is especially true if the leucocytosis is wanting when the patient is critically ill. With actual leucopenia, or the condition now known as agranulocytosis, the conditions are even more serious and the mortality is very high.

A boy (H. A.), age 13 years, became ill in March, 1929. He was attended by the family physician for two weeks, during which time he ran a temperature as high as  $104^{\circ}$ . He had considerable nasal obstruction and discharge, and also a very sore throat.

The condition was called "influenza," and the child seemed to be making a normal recovery until he developed a severe ache in the right ear. Because an operation for acute mastoiditis on this side had been made four years previously, the family became alarmed and called for an otologist. The right ear drum was found red and bulging. Paracentesis was done at once, and the temperature, then  $103.5^{\circ}$ , dropped in forty-eight hours to normal. The ear discharged a serosanguinous fluid profusely for four days. There was no tenderness, redness or swelling in the scar of the mastoid operation during this time. The left ear also became involved, was also incised and ran the usual course of an acute otitis media. On the fifth day slight tenderness developed in the scar of the operation four years before, and the temperature rose to  $102^{\circ}$ . The following day there was slight redness in the scar, and the patient was admitted to the Michael Reese Hospital. Admission temperature,  $100.4^{\circ}$ ; pulse 112; hemoglobin, 80 per cent; leu-

cocytes, 15,900, with neutrophiles 78 per cent, small mononuclears 14 per cent, large mononuclears 7 per cent, transitional 1 per cent, and no abnormal forms. Fluctuation was now apparent in this scar and the mastoid was again opened. In it was a large cavity filled with pus and lined with granulation tissue, and bordering the cavity a few small remaining mastoid cells were found and exenterated. On the following day the fever rose to  $104.2^{\circ}$  but gradually dropped to normal on the seventh day. Healing was progressing nicely and he was discharged three days later. But that evening he had a severe chill, the temperature mounted to  $104^{\circ}$  and he was readmitted to the hospital. The leucocytes, which on the sixth day were 9,800, were now only 10,700, though the temperature ranged from 101 to  $104.8$  degrees. Efforts to secure cultures of bacteria from the blood failed. The eye grounds and urine were normal. The serum failed to agglutinate typhoid and paratyphoid bacilli.

On the fourteenth day after his return to the hospital the temperature was  $104.8^{\circ}$ . He had one slight and one severe chill. The leucocytes at this time numbered only 6,000, with neutrophiles 42 per cent, small mononuclears 55 per cent, large mononuclears 2 per cent, transitionals 1 per cent and no abnormal white cells; red cells, 3,450,000; no malarial parasites in the blood. The right lateral sinus of the dura was exposed, appeared normal and was not opened. The next day the leucocyte count was 7,500, but the general condition of the patient seemed less favorable, and he was given a transfusion of whole blood, 250 cc., by the direct method. The fever of  $103.4^{\circ}$  at the time of transfusion dropped to below  $100^{\circ}$  the following day and was normal two days later. On the fifth day after the transfusion, and the nineteenth day after this second mastoidectomy, the temperature rose to  $100.6^{\circ}$ , where it remained three days; the leucocyte count was now 11,500; red cells, 4,200,000; neutrophiles, 65 per cent; small mononuclears, 21 per cent; large mononuclears, 12 per cent, and transitionals 2 per cent. At noon of the twenty-fourth day he had a severe chill lasting 20 minutes. From blood taken 24 hours previously a hemolytic streptococcus was grown. The right external jugular vein was now ligated and the right lateral sinus opened and found thrombosed, but the clot was easily removed and

free bleeding obtained. A pack was inserted into the mastoid cavity and other usual postoperative procedures instituted. On the following day he was given another transfusion of 340 c.c. of blood from the father. On this day the temperature did not exceed 102.4°, but it was 104.8° the next day. This was the last flare up of fever, for it never was over 100° after that. He was discharged on the forty-third day after his first admission.

In the blood at the last examination there was 80 per cent Hb; reds, 4,200,000; leucocytes, 9,200, with nothing of significance in the differential count. During this protracted illness, during which the mastoid was opened three times, there was no delay in healing. When he left the hospital, the mastoid incision, the jugular incision and that made to transfuse blood into the left cubital vein were all well healed.

It may appear that there was very little relation in this prolonged mastoid infection to diseases in which agranulocytosis has been observed. But we became interested in this possibility, and at one time during the illness alarmed when the state of the leucocytes obviously indicated so little of the customary systemic response to infection. This brought about the brief summary of prevailing conceptions of agranulocytosis with which somatic defensive measures are so wanting that death frequently results. The suggestion is also made that at one time in this patient H. A., a much less but in other particulars similar lowering of resistance occurred during his obstinate mastoid infection. Careful studies of the blood should be made early and continued throughout the course of all serious acute localized infections of the nose, throat and contiguous regions. From such studies marked impairment of immunity reactions will occasionally be discovered, and eventually means will be found to correct such defects and obtain the help so essential for successful surgical intervention.

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## XII.

### THE CAUSE AND TREATMENT OF CICATRICAL STENOSIS OF THE LARYNX.

BY MILLARD F. ARBUCKLE, M. D.,\*

ST. LOUIS.

In a paper on stenosis of the larynx, published in the Archives of Oto-Laryngology, May, 1929, by Professor E. Schmiegelow of Copenhagen,<sup>1</sup> this author states that fifty years ago every case of chronic stenosis of the larynx had to be considered incurable. He also says that modern laryngologists may claim that every patient with chronic stenosis due to cicatrization of the soft parts, syphilis, perichondritis, ulceration, post-operative lesions, diaphragmatic formation, synechia and similar pathologic process, can be cured, etc. This article is most instructive and stimulating, discussing as it does the older methods of treatment, the gradual evolution of the present methods of dealing with these cases and finally discussing clearly and convincingly his own method of treating chronic stenosis of the larynx.

Sir St. Clair Thomson<sup>2</sup> says the cure of stenosis of the larynx is notoriously unsatisfactory.

Dr. M. Paunz,<sup>3</sup> in a paper published in the Zeitschrift Hals Nasen und Ohrenheilkunde, January, 1929, 22:424-439, describes fully the etiology and pathology of diphtheritic stenosis and his method of treatment. He emphasizes the harmful effect of high tracheotomy and improper use of the intubation tube. In cases in which all other measures have failed, he recommends laryngofissure, both as a method of establishing an exact diagnosis of the condition present, as well as affording an opportunity for applying proper treatment. He favors removal of the scar tissue and then placing an iodoform gauze plug in the lumen and allowing this to remain five or six days.

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He then uses rubber or metal plugs for a few weeks until epithelization takes place.

Dr. G. Lange,<sup>4</sup> in a paper published in the *Zeitschrift for Laryngology and Rhinology*, 1929, 18:95-100, advocates dissecting out the scar tissue submucously, replacing the fragments of mucosa in the normal position and keeping down exuberant granulations by frequent bouginage.

Dr. A. Rethi,<sup>5</sup> in the *Zeitschrift Hals Nasen und Ohrenheilkunde*, 15:315-317, October 6, 1926, describes his difficulties with collapse of the voice box after Thiersch graft had been placed in position, because of previous cartilage loss.

This same author,<sup>6</sup> in *Zeitschrift for Laryngology and Rhinology*, advocates removal of scar tissue from the lumen and the use of metal obturators to permit healing without contraction.

Dr. Samuel Iglauder<sup>7</sup> has described in the *Transactions of the American Laryngological Association*, XI, 1918, page 329, a method of applying intralaryngeal pressure by means of a folded rubber tube. This procedure will undoubtedly be of great assistance in reestablishing a normal lumen when enough lumen remains to permit placement of the rubber catheter.

It is my purpose to call attention to the most frequent causes of cicatricial stenosis of the larynx, to discuss the possibilities of avoiding it, and to describe the methods now employed in treating these cases.

The chief cause of necrosis of the laryngeal tissues, of course, is infection. Diphtheria and accompanying mixed infection is the most frequent cause. Some European authors place syphilis first among causes. Infections with the streptococcus, staphylococcus, influenzal bacillus, typhoid bacillus, tubercle bacillus, smallpox, measles, whooping cough and numerous others may also cause necrosis of the laryngeal tissues. Trauma from external injury, such as gunshot wounds, falls, blows, suicidal attempts, etc., followed by infection may be a cause. Trauma during the course of treatment of laryngeal diphtheria or any form of acute obstruction of the larynx may, and frequently is the cause of chondritis and perichondritis with subsequent stenosis. Trauma may come from the intubation tube or from a tracheotomy tube. When from the intubation tube, trauma sometimes is inflicted when efforts are

made to place the tube in position. This point was fully covered by H. L. Lynah,<sup>8</sup> in a paper published in the *Laryngoscope*, September, 1918, 28:629. Pressure points at the level of the cricoid cartilage, the lower border of the thyroid cartilage, on the lateral wall just below the true cords, on the cords or on the false cords, will cause tissue death if not relieved promptly. It is almost certain to have occurred by the end of three days of continuous pressure. I subscribe fully to the use of the intubation tube in laryngeal diphtheria with embarrassed respiration, but I am convinced that in many cases the tube is coughed up and replaced far too many times for the good of the patient. In other cases the tube is allowed to remain in position much longer than is well for the laryngeal tissues. When a tube is constantly being coughed up and replaced, or when decanulation cannot be safely carried out at the end of five or six days, I believe a low set tracheotomy should be done. St. Clair Thomson recommends removal in three days. If continued, replacing of the tube becomes more difficult at each sitting, with increasingly greater trauma, greater strain on the patient's heart and greater danger to life, either by suffocation or heart failure. Under such conditions an emergency tracheotomy nearly always becomes necessary, and this usually is not so well done as is a set tracheotomy. Emphasis is laid on the statement that a low tracheotomy should be done in order to avoid pressure to the cricoid cartilage. Pressure on the cartilage of the larynx by a tracheotomy tube placed too high is one of the most frequent causes of laryngeal stenosis. Since cardiac involvement is so frequent and severe in late cases of diphtheria, freedom from struggle or any form of muscular exertion on the part of the patient is of utmost importance. Notwithstanding the general feeling that the mortality after tracheotomy in diphtheria is high, it has been my experience and is my belief that the high mortality rate might be lowered somewhat if the tracheotomy were done earlier in the course of the disease. An early tracheotomy in such cases is as nothing compared with what must be endured by these unfortunate individuals once necrosis and subsequent stenosis has occurred. Frequently, individuals suffering with postdiphtheritic stenosis are diphtheria carriers and are a source of danger to others until the infection is

cleared up. This usually is accomplished only when the scar tissue has been removed, and this may be a matter of months or years.

The treatment of the different types of cicatricial stenosis varies. For example, the so-called supraglottic hypertrophic type without involvement below the cords is comparatively easy to deal with. In these, the scar tissue can be removed with instruments and with the electrocautery, and resorption further encouraged by bouginage. Free use of the voice apparently promotes resorption and the formation of new cords if these have been involved. Webs across the lumen may be removed by cutting and dilating, but they have a tendency to form again unless frequent bouginage is carried out.

When the perichondrium and the cartilage have been destroyed there is greater disturbance in the arrangement of the framework of the larynx, and a more marked stenosis occurs than is seen after superficial necrosis. It has been in this type of case that I have experienced the greatest difficulty when treatment was undertaken. In these which I have seen, the cartilage of the left thyroid ala in about its lower three-fourths, and that of the cricoid in its left side almost one-half around had apparently been destroyed by necrosis. Also, there had been superficial necrosis in the region of the cricoid on the opposite side and apparently around the remaining portion of the ring inside. Scar tissue had formed and by contraction had dragged the left wall across the lumen of the larynx, past the midline, and a very firm attachment, forming sort of an oblique diaphragm without an opening, was thus established across the lumen of the larynx. Inquiry brought out the fact that in each case intubation had been done repeatedly over a long period with increasing frequency. When intubation became no longer possible, emergency tracheotomy was done. I was interested in finding destruction of the cricoid and thyroid cartilage in these cases only on the left side. Further investigation brought out the fact that all intubations had been done by a right-handed man. This is mentioned because of my belief that trauma is an important factor in the cause of tissue loss. In these cases no lumen remained. Efforts at passing instruments by endoscopic measures were without avail. When the larynx was opened it was found that the site

of the former lumen was occupied by scar tissue containing islands of cartilage.

In the past, laryngostomy has been employed with success in the treatment of this type of case but it is a most tedious and long drawn out performance. Professor Schmiegelow first does a low tracheotomy, moving the tube away from the stenosis as far as possible. After proper time has elapsed, probably two months, he does a laryngofissure, exposing the lesion to direct observation. After dissecting out the scar tissue and reestablishing the lumen, he fills this trough with an india rubber drain and closes the larynx, holding the rubber in position by means of silver wire through and through, cut off close to the skin and allowed to remain in position for a considerable period. This may be weeks or months. He reports most satisfactory results.

In view of Gillies<sup>10</sup> experience, in which he showed it was possible to prevent contraction of reconstructed noses by supplying proper lining, it occurred to me that it might be possible to reconstruct and reline the larynx,<sup>11</sup> thus preventing contraction. I have done this in four cases, two of which are living after a period of three and four and one-half years, respectively, without recurrence of stenosis; one died a few months after operation from peritonitis without recurrence of stenosis, and in the fourth we have thus far failed, although two attempts have been made.

In this procedure, after all other methods of reestablishing the lumen had failed, a laryngofissure was done and the scar tissue cleared out of the lumen, leaving a trough as in laryngostomy. A piece of sea sponge cut to fit this trough snugly was covered by Thiersch graft and placed in position. The larynx was closed by layers, but before closure a through and through silkworm stay suture was placed in position. If the stay suture is not employed, the obturator will surely be coughed up. At the end of eight days the stay suture was cut and the sponge withdrawn by direct laryngoscopy. No further treatment has been necessary in these cases. In the fourth case, a child, aged four years, vomiting occurred during the operation, and while we all know a Thiersch graft will take in the presence of infection, this one did not do well. At a

subsequent operation the stitches again sloughed out. There remained, however, sufficient lumen to admit passage of a small bougie.

At about this time, in view of his satisfactory experience in the treatment of cicatricial stenosis of the esophagus with electrically heated bougies, Dr. L. W. Dean suggested that this and two other highly refractory cases of laryngeal stenosis, in which a small lumen remained, be treated by this method. The bougie is heated to 40 degrees centigrade and left in place for thirty minutes. Dilatation by the usual process is then carried out. This work has been done by Dr. Wm. H. Johnston<sup>12</sup> and is reported under the title "The Use of Electrically Heated Bougies in the Treatment of Postdiphtheritic Cicatricial Stenosis of the Larynx," in the *ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY*, dated December, 1929. Dr. Johnston and all of us are very pleased with the results which he has obtained in these long standing and, to say the least, most discouraging cases. By application of this treatment at intervals of four or five days, over a period of eight to ten months, the lumen of the larynx is steadily increased in size, with corresponding improvement in the voice and ability to breathe through the natural passages. Improvement was apparent almost after the first treatment. This treatment has been employed in my case in which Thiersch graft failed with very considerable increase in the lumen of the larynx. I have no doubt that this will prove to be a most important adjunct in the treatment of lesions of this nature.

In connection with the operation of laryngofissure in children it has been said that development of the cartilages would be arrested by this procedure. In so far as I have been able to observe by seeing these cases from time to time since operation the growth of the larynx is keeping step with other parts of the body. ) /

My remarks concerning the etiology in these cases are based on impressions gained by observation during treatment, as well as the hospital records and histories otherwise obtained.

It is quite obvious that the method of restoring the lumen, employed in the cases with total atresia, was attempted only as a last resort. Also that more experience is necessary before we may speak with any great feeling of assurance. It is

equally unnecessary to remind those doing this work of the possibilities, if we may be enabled more often to prevent the occurrence of stenosis, and when present to expedite the treatment.

#### CASE REPORTS.

C. G., age 16, female, admitted to St. Louis Children's Hospital, January, 1914, age 8 months. Diagnosis, laryngeal diphtheria with marked dyspnea and cyanosis. Antitoxin was given and intubation was done. The tube was repeatedly coughed up. Extubation was necessary in order to remove dried secretion over a considerable period. In October, 1914, emergency tracheotomy became necessary because of inability to replace the intubation tube, and for five years she was a diphtheria carrier. In 1916, direct laryngoscopy showed the opening of the larynx to be about  $\frac{1}{2}$  mm. in diameter with white scar tissue extending downward. In 1920, the tracheotomy tube was accidentally dropped into the trachea and was removed by bronchoscopy. I first saw this patient August 14, 1922. At this time there was very marked supraglottic hypertrophic cicatricial stenosis of the larynx. The scar tissue was piled up above the larynx, resembling somewhat the pictures seen of the old fashioned beehive. There was a small circular opening less than  $\frac{1}{2}$  mm. in size near the top of the mass of scar tissue. None of the normal landmarks of the larynx was visible. Treatment, repeated every five to seven days, consisted of removal of the scar tissue with the cautery and with punch forceps and dilatation by bouginage. This extended over a period of about two years. Apparently the opening increased in size, and the quality of the voice improved as ability to use the larynx increased. The tracheotomy tube was removed about two years after I started treatment and has not been required since that time. She has quite a serviceable larynx for both respiration and phonation, although her voice is somewhat husky. The right vocal cord appears to be normal, but there is slight roughening along the edge of the left cord. I think there is also some restriction in movement of the left arytenoid cartilage. However, there is practically no sign of the mass of hypertrophied scar tissue above the larynx. A small fistula remains at the site of the tracheotomy wound. This will be closed by plastic operation later.

This case is a beautiful example of the serious damage to the larynx which may occur when a tube is worn longer than it should be. It also demonstrates the possibility of recovery of the laryngeal functions in cases of supraglottic hypertrophic cicatricial stenosis, even in the presence of the severest type of lesion.

H. C., age 11 years, female, came because of interference with breathing. She had stridor and cyanosis. Her symptoms were worse at night. Otherwise she was healthy. At the age of about two years, patient had diphtheria and wore an intubation tube for a long period. It evidently was a severe diphtheria, since she had paralysis of the soft palate afterwards. Indirect laryngoscopy revealed a web occupying about seven-eighths of the lumen of the larynx at the level of the cricoid. The remaining opening was oval shaped. Under direct laryngoscopy the web was split and dilated. It was dilated three times, after which the patient disappeared for about four years, at which time the mother came in for treatment, and the child was brought in by request in order that I might examine her. On questioning, I find that she is very fond of dancing and violent exercise. When she dances there is a slight inspiratory stridor but not enough to interfere in any way. There is slight thickening in the region of the cricoid which might not be noticed if one were not looking for it. There was no injury to the vocal cords at any time. Development of the larynx is normal so far as size is concerned.

A. B., age 3, male, admitted to St. Louis Children's Hospital March 3, 1929. No history obtainable. He had marked stridor with cyanosis, retraction and a bloody nasal discharge. Diagnosis, nasal, pharyngeal and laryngeal diphtheria. Antitoxin was given immediately and intubation was done at once, but the tube was soon coughed up. Within the next three days he was intubated ten times. On March 7th, emergency tracheotomy was done. On March 20th, attempt to reduce the size of the tracheotomy tube resulted in asphyxia severe enough to necessitate artificial respiration. On the 30th, he was unable to breathe without the tube. On April 5, 1929, by direct laryngoscopy, stenosis was found below the cords. At this sitting and on the 13th, I was unable to pass a No. 16 dilator or a 3 mm. bronchoscope. On May 4, 1929, the larynx was opened

by laryngofissure. When the larynx was opened the cause of stenosis was found to be a plate of dense scar tissue extending from the left side about midway of the thyroid cartilage downward and obliquely across to the right side where it was attached at the level of the cricoid. This scar was surprisingly dense for the duration and completely obstructed the lumen of the larynx. There was some deformity of the left ala. The obstructing scar tissue was dissected out and the denuded area was covered by mucous membrane graft taken from the upper lip. The graft was prepared according to the description above. Vomiting into the wound occurred during operation. The larynx was closed in layers and a stay suture anchoring the graft was used. On May 13, 1929, the sponge was removed by direct laryngoscopy and the 4mm. bronchoscope passed readily. At this time it was evident that the graft had not been kept in apposition to the denuded spot since operation. June 1, 1929, there was obstruction to breathing. I was then unable to pass a 3 mm. bronchoscope, and a No. 16 dilator was passed with difficulty. June 7, 1929, dilatation by direct laryngoscopy. July 5, 1929, dilatation was done but it was apparent that obstruction was increasing and by this time was quite marked.

July 15, 1929, laryngofissure was again done and Thiersch graft was applied.

August 20, 22 and 29, 1929, dilators ranging from sizes 16 to 20 were passed, but it was apparent that contraction was present and increasing.

September 1, 1929, was unable to breathe through larynx.

September 3, 1929, at the suggestion of Dr. L. W. Dean, treatment by electrically heated bougies was instituted every three to five days. It was apparent almost at once that improvement was being obtained. Dilators were increased from one to two sizes at each sitting. September 27, 1929, he could cough through his larynx but was still unable to breathe through it.

October 14, 1929, he was getting some air through the larynx. At the present time his voice is good enough that he calls the nurse from the ward when she is in her office, which is around two or three corners and a distance of about 45 feet. He can breathe quite comfortably through his larynx and goes as long as twenty minutes without the tube. There is, however, a very evident neurotic element (fear) present in this



case, and it is my feeling that this has a very important bearing on the length of time during which he may be without the tube. I think the reaction to the heated bougies has demonstrated in this case that it is a very important step in methods of causing resorption of scar tissue. It is, of course, apparent that the skin graft operation failed in this case from the standpoint of securing sufficient lumen. From the findings at the time of operation, however, it is also quite evident that bouginage was out of the question until some lumen had been afforded. Because of the position of the obstructing scar tissue, its texture and its firm adhesion to the walls, it is my belief that laryngofissure was probably the safest approach for the purpose of opening the lumen.

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### XIII.

#### RUPTURE OF ABSCESES INTO THE EXTERNAL AUDITORY CANAL.

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PHILADELPHIA.

The rupture of an abscess into the external auditory canal is a very uncommon occurrence, if one can judge by the number of cases reported in the literature. Very little mention has been made of such conditions. While there has been practically no formal recording of the rupture of abscesses into the external auditory canal, there has been a number of cases mentioned in the discussions of various articles, especially those pertaining to retropharyngeal abscess. Therefore, it would seem that the condition is more common than we are led to believe. It might be well to report more of such cases, for the development of such abscesses give not only an interesting picture but also one that may present very definite difficulties of diagnosis and some difference of opinion as to the treatment.

#### LYMPHATIC SYSTEM OF THE NECK.

The study of such a condition must fall most prominently on a study of the lymphatic system of the neck, as abscesses in the vicinity of the external auditory canal are apt to be primary in these lymphatic glands.

Wood<sup>1</sup> divides the cervical lymphatic nodes into two main groups, the superficial or collecting nodes and the deep or terminal nodes. The superficial group is composed of the following subgroups:

1. Suboccipital group and aberrant glands of the nape of the neck.
2. Mastoid group.
3. Parotid and subparotid group.

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4. Submaxillary group with the facial glands as an offshoot.
5. Submental group.
6. Retropharyngeal group.

All of these subgroups except the submaxillary and submental must be considered as possible sources of infection that may spread to the auditory canal, because they all have efferent lymph vessels that empty into the highest glands of the deep lateral chain. The suboccipital group receive lymph vessels from the back of the head. The mastoid group, usually two in number, drain the temporal portion of the hairy scalp, the internal surface of the auricle, except the lobule, and the posterior surface of the external auditory meatus. The parotid group receive afferent vessels from the external surface of the auricle, from the external auditory meatus, from the tympanum, from the skin of the temporal and frontal regions and possibly also from the eyelids and base of the nose. The subparotid lymph glands are beneath the parotid salivary gland, between it and the pharyngeal wall in the lateropharyngeal space. They drain the nasal fossæ, the nasopharynx and eustachian tubes. The retropharyngeal glands receive their afferents from the mucous membrane of the nasal fossæ and accessory sinuses, from the nasopharynx, from the eustachian tube and probably from a part of the tympanic cavity.

The descending cervical chain of lymph nodes consists of two sets of glands: the deep cervical chain and several more superficial chains. The deep nodes extend from just beneath the ear downward under the sternocleidomastoid muscle, generally only as far as the point where the omohyoid crosses the vessels and nerves, but occasionally reaching as far as the junction of the internal jugular and subclavian veins.

It is these deep lymph nodes and the retropharyngeal group that are probably mostly concerned in the production of abscesses that may rupture into the external auditory canal. The retropharyngeal glands lie in the tissue behind the pharyngeal wall and in front of the deep prevertebral muscles at the level of the upper two or three cervical vertebræ. These glands are arranged in two groups on each side; one group in the median position and the other more laterally situated. These median lymph glands are especially well de-

veloped in the first year of life. Thereafter they undergo progressive atrophy, and when adult life is reached have practically always disappeared. The laterally situated glands are usually two in number on each side, arranged vertically. This group is present in infants, usually persist into adult life and may give rise at any time to a retropharyngeal abscess. According to Most, the lymphatic drainage from the nose and throat to the retropharyngeal glands is only minor, the principal drainage from the upper respiratory passages being directly to the deep cervical glands.

It can be seen how easily possible it would be for one of the common infections of the throat or sinuses to infect one or more of the highest placed of the deep cervical lymph glands, either directly or through the retropharyngeal glands, and to have the resulting abscess rupture into an adjacent structure such as the external auditory canal. As a matter of fact, a number of cases in which a retropharyngeal abscess has ruptured into the canal have been mentioned, usually in discussions on the subject of retropharyngeal abscess.

Mr. A. Cheatele, in discussing a paper by McKenzie,<sup>2</sup> on otitic pharyngeal abscess, briefly mentioned three cases of abscesses breaking into the external auditory meatus. The first was a specimen he found postmortem, in which there was an abscess lying behind the jaw and a carious opening in the meatal wall, leading to the abscess. The second case was operated upon and the anterior meatal wall was found replaced by a granulating hole through which a finger could be passed into a large abscess cavity behind the jaw to the tonsil. His third case was one in which trouble in the anterior meatal wall led to an abscess which pointed into the pharynx, where it burst. Fulkerson<sup>3</sup> vaguely mentioned cases of Hessler, Kapler and others in which fistula occurred in the anterior meatus, resulting from an abscess in the pterygoid or pharyngeal region or diseases of the pneumatic cells in the anterior meatal wall or defects therein, the result of which is an ulceration into the external auditory canal. Fulkerson does not go into any more detail of these cases than as above quoted, and a search of the literature did not reveal them.

Eves,<sup>4</sup> in 1926, reported a case of retropharyngeal abscess discharging through the right external auditory canal. The

patient was a child, twenty months old, who was admitted to the hospital with extreme dyspnea and cyanosis. A short time later these symptoms were relieved with the coincident discharge of pus from the right ear. This was later proved to be a retropharyngeal abscess that had evacuated itself through the anterior wall of the right external auditory canal at the junction of the cartilaginous and osseous portions.

Eves mentioned that there were three routes this pus could have followed to reach the auditory canal. (1) Through the fissures of Santorini; (2) through the fibrous tissue between the junction of cartilage and bone, which is at times present, especially in children; and (3) through a dehiscence of the bony canal, which is present in a small percentage of cases. In the discussion of this paper, Dr. George B. Wood cited a case of quinsy which evacuated through the external auditory canal, and Dr. N. P. Stauffer mentioned a similar case of retropharyngeal abscess discharging through the ear.

Such meager reports as the ones above recorded have from time to time appeared. No doubt many others have been observed and not reported. There appears to have been a lack of interest in such cases, which is not warranted when one considers the fact that such a case as the one following can present not only some difficulty in differential diagnosis but also may require keen judgment and some surgical skill for its relief.

#### REPORT OF CASE.

The patient, a young woman, 28 years of age, first noticed pain under the left ear, just in front of the mastoid, on April 25, 1929. For a week this pain gradually increased, but daily examination failed to show any trouble whatever with the ear drum, canal or mastoid process. There was increasing tenderness just anterior to the mastoid tip but no swelling, redness or edema. During the second week there was the gradual development of a furuncle in the concha of the ear, which was incised with drainage of pus. This relieved the pain and tenderness in the auricle but had no effect on the postauricular pain and tenderness which had been steadily increasing. The temperature during this time had ranged from 101° to 98° F.

She was admitted to the surgical service of the University

of Pennsylvania Hospital on May 11, 1929, the pain by that time having become almost unbearable. Examination on admission showed the eyes, nose and throat entirely negative. The tonsils were absent, having been removed several years before. The anterior and posterior cervical lymph glands were slightly enlarged and tender, and the area under the ear was so tender that detailed examination could not be permitted. The ear drum and canal were entirely negative, but there was slight edema of the auricle. Blood count showed 12,100 leucocytes.

The previous medical history was unimportant except for the fact that she had had a prolonged cold suggestive of a sinus infection, which had cleared up at the time of admission.

Examination on May 13th, two days after admission, showed an increase of the edema of the auricle and for the first time a slight swelling of the posteroinferior wall of the cartilaginous canal. The drum was still negative. Dr. E. P. Pendergrass gave the following report on a roentgenogram of the mastoid process: "There is a slight haziness of the cells in the outer part of the petrous portion of the temporal bone of the left side but no definite evidence of breaking down of cell walls."

On the next day there was still more edema of the auricle and also edematous swelling in the preauricular region. The lower posterior canal wall showed an increased swelling, and the tender area below the ear now showed some evidence of fluctuation but only a slight swelling. The high temperature for the day was 100.6° F. The leucocyte count was 12,500.

On the following day there was a spontaneous rupture of this abscess through the posteroinferior wall of the cartilaginous canal with the evacuation of a large amount of pus. This gave considerable relief of the pain below the ear for a short time, but within a few hours the pain and tenderness had again increased. Therefore, on the next day, May 16th, Dr. E. L. Eliason opened the abscess just posterior to the auricle. A note by Dr. J. P. North concerning the operation reads as follows: "An incision was made just anterior to the left mastoid process paralleling the ear. The tissues were rather diffusely necrotic and thick yellow pus escaped from a cavity which extended for a depth of at least an inch just anterior to the border of the sternomastoid muscle near its

origin. This cavity communicated with the opening through the posterior wall of the cartilage of the external auditory canal. Extensive exploration was not done because of the fear of injuring the facial nerve. Pus could be expressed from the tissues of the neck below the mastoid tip, so the incision was prolonged downward for a short distance. One piece of rubber dam and a small piece of gauze were used for drainage."

Following this operation there was immediate relief of the pain, and the swelling of the face, neck and auricle slowly disappeared. Considerable pus drained from the postauricular wound for about two weeks, when the opening closed. During this time pus drained constantly from the auditory canal in a lessening amount. As soon as the postauricular wound ceased draining there was an increase of pain, which was again relieved by the pus rupturing through the healing incision. There was drainage through this incision for the following three days, when the wound again closed. During these three days there had been no discharge from the auditory canal, but on cessation of the drainage externally there appeared a swelling in the canal wall. This was opened with the evacuation of pus.

During the next two weeks it was necessary to incise the posteroinferior wall of the canal twice under gas to allow the accumulated pus to escape. Following this there was a constant profuse discharge of thick pus through the canal wall for several weeks. On July 17th, two months after the operation, while probing the opening through the canal wall, an irregular flat piece of bone, about 7 mm. long and 4 mm. wide, was dislodged and removed. A roentgenogram four days previous to this showed the mastoid process negative and no evidence of necrosis of the posterior wall of the left auditory canal. However, because of the finding of the necrosed piece of bone the patient was sent to the hospital, and under gas a thorough curetment of the opening in the auditory canal was done. No other loose pieces of bone were discovered.

From this time on recovery was rapid. The purulent discharge through the canal wall promptly stopped and there was a rapid filling in of the curetted cavity by granulation tissue, which later became covered over by a skin surface, leaving only a slight depression in the wall of the canal.

## COMMENT.

This patient presented some interesting and puzzling features. The development of the abscess gave symptoms referable only to the ear and mastoid process, but repeated examinations of the auricle, canal, drum and mastoid process were entirely negative. As the nose and throat were also negative a diagnosis became difficult to make. The intense pain and tenderness in front of the mastoid tip, of course, suggested the development of an abscess, but it was so deep seated that no real swelling or fluctuation appeared at the surface. A mastoid abscess, while possible, was not seriously considered with the absence of any middle ear trouble at any time and with a practically negative roentgenogram.

After the abscess had ruptured into the external auditory canal and later had been freely drained by incision behind the auricle it was expected that the discharge would rapidly subside. The drainage, however, persisted for several weeks and there was some suspicion of necrosis of cartilage. It had been decided to detach the auricle posteriorly and search for necrotic cartilage when the piece of bone which had sloughed through the canal wall was discovered. This bone apparently came from the osseous portion of the canal wall, but there was no evidence of it from the roentgenogram taken a few days previously.

The cause of the abscess and the exact spot where it originated have not been definitely decided. It has been assumed that it originated in one of the highest placed lymph glands of the deep cervical chain. Perhaps one of these glands became infected through the lymphatic drainage from a sinus infection which the patient probably had. The abscess that subsequently developed followed the line of least resistance, which, due to its being so deep seated, happened to be into the external auditory canal.

A review of such a case as this can give rise to considerable speculation as to the cause, method of extension and origin of such an abscess in addition to presenting interesting features of diagnosis. It is hoped that reports of similar cases will be forthcoming, which will eventually lead to a more complete understanding of such conditions.



## SUMMARY.

1. The rupture of abscesses into the external auditory canal has been but little recorded in the medical literature.

2. The study of such conditions must fall most prominently on a study of the lymphatic system of the neck, as abscesses in the vicinity of the external auditory canal may be primary in these glands.

3. Report is made of a case of rupture of an abscess into the external auditory canal, apparently originating in one of the highest placed lymph nodes of the deep cervical chain.

4. Such cases present such interesting features and such grounds for speculation as to the cause, origin and method of extension that it would seem essential that details of similar cases should be reported so that we can arrive at a more complete understanding of such conditions.

2117 CHESTNUT ST.

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#### XIV.

### TYMPANOGENOUS LABYRINTHITIS RESULTING FROM AN ACUTE OTITIS MEDIA IN ITS EARLY STAGES, OR IN ITS SEROUS STAGE: REPORT OF TWO CASES.

BY ERNEST M. SEYDELL, M. D.,

WICHITA.

Labyrinthitis arising during the first few days of an acute otitis media is an uncommon complication. In the two cases which I shall report, both patients were women. One was 55 years of age, the other 56 years. Both were the wives of physicians and each had a son, who also was a physician.

I do not intend, in this paper, to completely describe the symptoms, pathology, prognosis and treatment of suppurative labyrinthitis, but will confine myself to a short outline of this subject, particularly as it relates to the cases reported.

An invasion into the labyrinth during the course of an acute otitis media usually takes place through the round or oval windows. The round window is the portal of entry in the majority of cases. There is still considerable difference of opinion in reference to the importance of the oval window as a portal of infection. The invasion may either be bacterial or may be due to a *Durchwanderung* or dialysis of toxins through the intact membranes. The majority of the investigators are of the opinion that the latter method of invasion is by far the most common.

The destruction of the membrane itself does not usually occur early in the disease.

Infection may also enter the labyrinth through the vascular communications that exist between the mucous membrane of the middle ear and the endosteum of the labyrinth. In rare instances, a dehiscence in the labyrinthian wall may be the point of entry.

In a general way, one may classify acute infections of the labyrinth as serous or purulent. Toxins, entering the labyrinth,

usually produce a serous labyrinthitis. If bacteria gain entrance into the labyrinth in the later stages of the middle ear infection, suppuration will usually supervene. In turn, the invasion may remain circumscribed or become diffuse.

Symptoms.—The onset is sudden. The labyrinth may become involved as early as the second day of the middle ear infection. Vertigo, nausea, vomiting and disturbance of equilibrium, accompanied by more or less prostration, dominate the clinical picture. Tinnitus usually adds to the patient's discomfort. Deafness of sudden onset, varying in degree, will also be found. Nystagmus, whose direction and degree are dependent upon the location and severity of the infection, is constantly present.

Headache is not a symptom of labyrinthian inflammation. When present, it is caused by some concomitant lesion or is due to an intracranial extension.

The same may be said of fever. There are, however, some exceptions to this rule, such as the cases reported by Bondy, Alexander and Lund.<sup>1</sup>

Treatment.—In no other complication of middle ear suppuration is the question of treatment so difficult to determine, and in no other condition does such a diversification of opinion exist.

In a short survey of the literature relative to the treatment of labyrinthitis, beginning during the serous stage of otitis media, I find much more unanimity of opinion than in any of the other types of this disease.

Bárány<sup>2,3</sup> is opposed to early operative interference, even in cases of diffuse suppurative labyrinthitis, and counsels waiting until the eighth day of the disease, when, if there is no restoration of function, a labyrinth operation should be performed.

Hautant<sup>4</sup> considers that conservative treatment is a safe procedure.

Zange.<sup>5</sup> Conservative treatment is the rule. He does not consider that a complete absence of reaction is an absolute indication for surgery, except in cases of influenza.

Holmgren<sup>6</sup> is of the opinion that the diffuse serous type, with considerable loss of function, must be as closely observed as the suppurative type, since both frequently terminate in

meningitis. Where no labyrinth function exists, he advises immediate operation.

Hinsberg<sup>7</sup> is very conservative. He operates on exceptional cases of the diffuse suppurative type.

Gunther<sup>8</sup> has the same opinion.

Ruttin<sup>9</sup> favors very conservative methods of treatment. He states that the earlier the invasion, the more certain it is to be serous, and consequently the more favorable the prognosis. A dangerous labyrinthitis may, however, arise during the early stages of a middle ear inflammation. He reports eleven cases of labyrinthitis, which began during the serous stage, and were treated solely by medical means. Of this number eight lived and three died.

Knick<sup>11</sup> is conservative, but is of the opinion that these cases are more dangerous than is generally supposed.

In a general way, the treatment of this type of labyrinthitis should be conservative.

The patient should be closely observed and kept as quiet as possible. All unnecessary movement of the head should be prevented. Frequent spinal punctures should be made, it being recognized that the results of spinal puncture are only of value when positive, and that it is not always possible to obtain the spinal fluid at the proper time, as the infection may spread to the meninges within a few hours. The functional ear tests must be carefully carried out.

After studying many case reports in the literature I have come to the conclusion that it is advisable to delay performing any surgery on the mastoid process as long as possible. Traumatism always favors a spread of the infection.

Each case must be decided on the basis of a careful analysis of the findings present.

I do not feel that the absence of reaction to the functional ear tests is in itself a sufficient reason to necessitate a labyrinth operation. There should be no delay in operating on the labyrinth where symptoms of meningitis arise. I would even advocate a labyrinth operation in those cases where the infection is carried directly through the labyrinth to the meninges.

In the latter, the labyrinth and the meninges become involved at practically the same time. The infection is usually

of high virulence, and, in my opinion, the prognosis is almost hopeless with either the conservative or radical forms of treatment.

SUMMARY.

1. An acute labyrinthitis may arise as early as the second day of an acute otitis media.
2. This complication may occur where the tympanic membrane has not ruptured or where but little change of the drum exists.
3. The treatment should be conservative, with watchful expectancy. Mastoid surgery should be delayed as long as possible.
4. A labyrinth operation should be performed when symptoms of meningitis arise.

CASE HISTORIES.

Case No. 1.—Mrs. E. T., age 55. Date, March 13, 1928.

Past history: No serious illnesses; general health good; no previous ear trouble.

Chief complaint: Dizziness, nausea and vomiting; pain in right ear. One week ago patient developed an acute sore throat. Three days later this was followed by slight pain in the right ear. The pain grew worse during the following two days. At 3 a. m., on the morning of the third day, the patient suddenly developed an intense vertigo, nausea, vomiting and severe shock. Her pulse was very weak and of poor quality. On the way to the hospital the ear started to drain.

GENERAL EXAMINATION.

Nervous system: Reflexes normal; no headache; no double vision; spontaneous rotary nystagmus of the third degree to the left; pupils reacted normally.

Lungs, heart, abdomen, extremities were negative.

Examination of nose, throat and ear: Slight acute rhinitis and nasopharyngitis. Tonsils slightly inflamed. No membrane present.

Examination of her right ear revealed a large central perforation. The discharge was very profuse and was mucopurulent in character. The left drum was normal. There was some tenderness to pressure over the right mastoid. Functional hearing tests checked with the noise apparatus revealed normal

hearing in the left ear but complete loss of hearing in the right ear.

The patient's temperature on admission was 97, pulse 96, respiration 20; white count on admission was 15,000. She was quite drowsy.

Temperature curve: The highest temperature during patient's illness was 100.4. This was recorded on her second day in the hospital. Subsequent to this her temperature never registered over 100 degrees F., with a subnormal temperature in the morning.

Repeated white blood counts were made, the cell count decreasing each day.

Smears and culture taken from the ear revealed a hemolytic streptococcus. Blood cultures were negative.

Repeated X-ray examinations of the mastoid were made. The density of the mastoid increased, the cell outline became more and more indistinct, and finally an area of destruction became apparent in the posterior portion of the mastoid.

Course and treatment: The vertigo, nausea and vomiting gradually subsided. The patient never acknowledged a headache. The discharge became more purulent; drooping of the posterior superior canal wall gradually developed.

The patient's head was fixed with pillows, and ice bags applied to the diseased ear. A simple mastoid operation was performed on April 4, 1928. There was complete destruction of the mastoid cells, and a large quantity of pus and granulation tissue was found.

The patient made an uneventful recovery. Subsequent functional ear tests revealed complete deafness in the right ear.

Case No. 2.—Mrs. R. A. M. Date, September 15, 1928. Age, 56.

Past history: Several abdominal operations. General health good. No previous ear trouble.

Chief complaint: Dizziness, nausea and vomiting. Pain in the right ear. About five days previous patient developed a severe sore throat. Forty-eight hours after the beginning of the sore throat she developed a pain in her right ear. The pain grew steadily worse, and during the last twenty-four hours it was very severe. She was extremely nauseated, dizzy and restless, and moved constantly in her bed.

## GENERAL EXAMINATION.

Nervous system: Severe headache; no double vision; pupils reacting to light and accommodation. Nystagmus to the left, mixed type, third degree. Positive Kernig. Exaggerated reflexes. Her mental condition was very dull. It was difficult to make her understand.

Lungs, heart and abdomen were negative.

Examination of nose, throat and ear: Severe acute rhinitis and nasopharyngitis. Tonsils inflamed; no membrane.

Right drum bulging; no discharge. Slight tenderness over mastoid. Left drum normal. Functional hearing tests checked with the noise apparatus revealed normal hearing in the left ear. Right ear showed complete loss of hearing; no reaction with cold water.

The patient's temperature on admission was 103.4 F. Pulse 104. Respiration 36.

Her white count on admission, was 18,000.

Temperature curve: On the afternoon of the admission into the hospital the patient's temperature reached 104.4. She had morning remissions. The lowest temperature recorded was 101, the average evening temperature being 103 F.

A smear and culture taken from the ear revealed the streptococcus mucosus.

Course and treatment: The ear discharged very freely after a paracentesis had been performed. The pus in the ear was under great pressure. Drainage of the ear did not relieve the headache and pain in the ear. A diagnosis of suppurative meningitis was made. A consultation with a neurologist confirmed this diagnosis. We were not allowed to withdraw any spinal fluid.

An unfavorable prognosis was made. There was no sagging of the posterior superior canal wall, and the tenderness over the mastoid receded.

The patient's condition grew steadily worse. Her neck became rigid. She became comatose and died four days after her admission into the hospital, or about nine days after her initial ear symptoms began.

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XV.

THE ROLE OF NASAL ACCESSORY SINUS MEM-  
BRANE IN SYSTEMIC INFECTIONS  
AND TOXEMIAS.

BY LAWRENCE J. LAWSON, M. D.,

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Clinical knowledge of the role of accessory sinuses as primary centers of infection has largely accumulated since the appearance, in 1882, of Zuckerkandl's "Normal and Pathological Anatomy of the Nasal Sinuses and Their Pneumatic Appendages." When compared to dental and pharyngeal infections undoubtedly the role is a minor one, but the aggregate number of instances of systemic disturbance from this source is considerable, and this possibility should never be overlooked. Clinical manifestations are largely those resulting from toxins, a smaller number are typically subinfections, and the remainder are a combination of these. It is the scope of this paper to consider: (a) factors altering the natural resistance of sinus membrane; (b) the physical and chemical qualities of the pathologic sinus content; (c) the probable point of passage of infectious material and toxins into the blood and lymph streams, and (d) to emphasize the inherent property of sinus membrane to return to an approximate normal when given physiologically proper drainage and aeration.

Normal sinus membrane is thin, pale and not vascular. For practical purposes it is histologically similar in the various cavities except in the ethmoid cells, where it is even thinner and more fibrous. It is distinguished from the general respiratory membrane by its thinness and by the scarcity of glandular tissue. The epithelium is composed of ciliated cells and is similar to that found in the respiratory portion of the nose. The subepithelial layer contains the blood vessels and scattered clusters of glands, and is intimately connected with the periosteum. Circulation takes place chiefly through the ostia, as would be expected from the embryologic origin of the sinuses,

although small vessels pierce the bony walls. The normal membrane offers considerable resistance to bacteria and toxins because of the normal mucus secretion covering it, the ciliary current, and normal aeration. Alteration of its resistance to infectious organisms is dependent upon a change of character of the mucus secretion, alteration or cessation of ciliary activity due to virulent bacterial toxins, or interference to normal ventilation. Suppurative inflammatory states are the most commonly recognized disturbances, but the nonsuppurative form of inflammation is a distinct entity and represents the majority of involvements. Theoretically, it always precedes suppuration and follows in the course of recovery after drainage or surgical correction. Because of the associated intrasinus involvement of the membrane in rhinitis, the term rhinosinusitis has been properly used.

In acute nonsuppurative inflammations the abnormal secretion of mucus, according to Zuckerkandl, is slight at first and increases only after hyperemia has existed for some time. The mucosa becomes gradually infiltrated and edematous, but the disorder is usually transitory and terminates in resolution.

When the disorder becomes chronic, exudation takes place, chiefly into the inner layer of the mucosa, the deeper periosteal cells become edematous and the whole membrane becomes thickened and often spotted with edematous elevations. This form of chronic nonsuppurative inflammation, according to Dmochowsky, may become hypertrophic or hyperplastic and transform the mucosa into a pale, hard membrane, a process which may almost obliterate the cavity or which may be arrested at any stage of the transformation.

In acute suppurative attacks the mucosa becomes hyperemic and edematous, with localized hemorrhages into the tissues, and its surface is bathed with pus. The changes are more rapid and severe with retention. According to many accurate observers, including Hajek and Zuckerkandl, the soft tissues are not swollen to the same extent in this condition as in the acute nonsuppurative form. An inflammatory exudate is produced, consisting of mucus, serum, leucocytes, exfoliated epithelium and bacteria. This is scanty at first, then serous or serosanguineous, then purulent. If virulent organisms enter,

the cilia are overpowered by bacterial and putrefactive toxins and swelling, and regions of punctiform hemorrhages and desquamation are produced. In the edematous membrane the intercellular spaces are filled with lymph, there is more or less round cell infiltration, there are punctiform hemorrhages through the connective tissue, engorged blood vessels but unchanged glands. The empyema usually terminates by resolution of the membrane, but if the process continues under unfavorable conditions of aeration and pressure, there is greater round cell infiltration, petechial hemorrhages, desquamation and occasionally the more serious pressure changes. Complete resolution cannot take place after this stage.

In the chronic suppurative state the membrane loses its cilia and becomes more or less covered with squamous or pavement cells. The glands are for the most part destroyed, and the blood supply is diminished by obliteration of the smaller arterioles and veins. The changes in the course of a purulent inflammation depend upon the duration of the disorder, the virulence of the attacking organisms and anatomic and drainage conditions. Because the blood vessels pass chiefly through the ostia, pressure in a given portion may cause localized swelling back of that location, which accounts for the circumscribed swelling and cyst formation, observed particularly in the maxillary sinus. In old chronic empyemas cheesy, fetid pus is frequently observed. In the hyperplastic type the membrane is often grayish, wrinkled, papillomatous and more or less loose from the underlying bone. Hyperemia if present is not marked and often localized. There may be edematous changes similar to nasal polyps, the connective tissue is thickened, and there are retention cysts caused by round celled infiltration constricting the necks of the glands. In the same specimen there may be atrophied glands and vessels or numerous new vessels due to new connective tissue. There are small areas of metamorphosis of ciliated to pavement epithelium. Osteoblasts may be found. There are seldom organisms in the mucosa at section.

The physiologic chemistry of the toxins contained and generated within the pathologic sinus is apparently but vaguely understood. Cholesterol is present in blood serum, according to Weston and Kent, to the extent of 2 grams per liter. Some

of the acids formed by the oxidation of cholesterol are poisons, having a toxic and hemolytic action comparable with some of the snake venoms. By repeated oxidation of cholesterol Windans obtained an acid more hemolytic than the bile acids. One of the acids was very powerful in dissolving red blood cells. The phosphatides are among the most important substances in living matter found in all cells, and undoubtedly function with cholesterol to produce the peculiar semisolid, semifluid state of protoplasm. The proteins are digestible by certain enzymes and on digestion yield amino acids. It has been suggested that substances having the property of raising the blood pressure may be produced from tyrosin in putrefactive processes and that these substances are active in causing arteriosclerosis. Putrefactive processes of this character may be factors in the production of degenerative changes and ageing. Much more accurate information and further experimental work are desirable.

With a knowledge of the changes accompanying inflammation an approximation may be made, with the aid of clinical observation, of the relative clinical importance of absorption of organisms and toxins directly through the sinus membrane and absorption in secondary locations after gravity and aspiration have distributed infectious and toxic material over more readily invaded tissues having greater absorptive qualities. The lower respiratory and gastrointestinal tracts may be affected by direct distribution along the membrane, by blood and lymph circulation or by inhalation of septic particles. Patients with active sinus drainage swallow great numbers of organisms with impunity, because the normal acid gastric secretion is an efficient antiseptic barrier. If mass infection persists it is suggested that potent toxins may render the gastric mucosa more easily attacked by invasion of organisms from the blood stream. Statistical studies seem to indicate that patients with active sinus drainage are more susceptible to attacks of cholecystitis and appendicitis. Most investigators believe, however, that the most likely route of invasion is through the blood stream. The production of severe constitutional symptoms, with high temperature, leucocytosis and severe adenitis is uncommon unless there is an accompanying secondary nasopharyngitis or other coincidental infection. A

Careful study of a number of office and hospital records of adult patients with acute suppuration does not reveal severe constitutional symptoms unless a severe infection accompanies or precedes the sinusitis. The temperature and leucocyte count are low, with a slight increase in polymorphonuclear cells. There is an absence of prominent cervical adenitis. It is only in the complicated, chronic sinus cases with edema, polypoid degeneration and putrefaction that noticeable systemic symptoms are produced. The common symptoms are usually those of a toxemia, such as a sallow skin with anemia, weakness and prostration, loss of appetite and lowered mental and physical efficiency. A toxemia does not necessarily mean a large dosage of absorbed toxin, for a virulent toxin requires but a trivial dosage. Very small amounts of toxin over a long period of time probably give origin to clinically important parenchymatous changes. It seems safe to conclude that there is relatively little loss of bacteria through the diseased membrane but that relatively small amounts of a virulent toxin when absorbed may produce disastrous results, particularly when continued over a long period of time.

Pickworth has presented specimens in a case of sphenoid sinusitis which, in his opinion, constitute a human parallel to Rosenow's experiments. A chronic diplococcal infection was traced invading the sinus mucosa from the surface and spreading through the pituitary capsule and gland. In the same patient similar diplococci were demonstrated in the deeper layers of hemorrhagic patches in the gastric mucosa. In this and other specimens he suggests that the diplococcal infection traced to the pituitary might explain the evidence of endocrine disturbance, since the superjacent hypothalamus is held to control certain metabolic processes. Elective affinity of certain strains of infection for certain tissues is supported by clinical observation, for one influenzal epidemic may involve the sinuses, another the lungs and another the gastrointestinal tract.

Mullin's experimental work required traumatism of the membrane before pigments and organisms could be absorbed, which demonstrated that absorption from the normal intact membrane is sluggish. He apparently demonstrated a lymph drainage through the submaxillary and deep cervical lymph nodes and ducts to the right side of the heart and the pul-

monary artery. Mosher states that the nasal sinus is an excellent place for the elaboration of toxins, even without demonstrable pus. Many so-called secondary infection syndromes attributed to disease of the sinus membrane may eventually be proven to be manifestations of toxemia or anaphylaxis. To illustrate characteristic toxic pictures, two typical reports are given.

Report No. 1.—A. H., aged 35, developed an acute left antrum suppuration following influenza two years before coming for examination. Antrum drainage continued, aggravated each time there was an acute rhinitis, these attacks coming at increasingly frequent intervals. Twelve months after the onset of the antrum infection there was noticed a lack of endurance, some pallor and loss of efficiency in his mental and physical efforts. In the course of a careful clinical examination a positive Wassermann was found, and the patient was put on specific treatment. Before appearing for examination intranasal antiseptic pack treatments, vaccines and gastrointestinal management had been undertaken with no improvement. When first examined a left pansinusitis was found. The left side of the nose was almost completely obstructed by swelling of the membrane. Ventilation was at first improved by an anterior middle turbinectomy, but the patient continued his toxic course, with severe headaches. One month later he was hospitalized and the sinuses of the involved side opened and aerated intranasally. Within two weeks there was a striking clinical improvement. When seen two months after the operative procedure the patient walked in an erect position, his disposition was decidedly better, he had returned to his normal neat appearance and had only a small amount of mucoid discharge, with no disagreeable odor. No secondary infections were revealed in any part of a thorough examination.

Report No. 2.—C. B. S., aged 30, developed an acute suppurative antrum infection following an influenzal attack three years prior to the time of examination. He was acutely ill at the time of onset, but soon improved in general health. The local pain and suppuration persisted in spite of frequent irrigations. The condition persisted for twenty-four months, during which time acute rhinitis attacks increased in frequency and each time increased the sinus drainage. Then a sallow color

replaced his usual ruddy complexion, there was loss of appetite, occasional tachycardia, increased drainage with a foul odor and weakness. Rhinologic examination at this time revealed a right pansinusitis. A septum resection and exenteration of the right ethmoid labyrinth was done. This produced only a slight improvement. When the patient again came under observation, nine months later, the maxillary and frontal sinuses were opened intranasally. Within two weeks the foul discharge cleared to one of clear, mucoid character, and the patient improved decidedly in strength. The patient when seen two months later was practically in normal health.

These two patients are examples of marked toxemia without demonstrable secondary infections. It is interesting to note that the tachycardia in the second case promptly subsided after improvement in the sinus membrane, and that a moderately increased blood pressure dropped ten millimeters after the original focus was aerated and drained. Literature on the relation of sinus involvement to the heart is scant. It is the opinion of the author, from his own experience and after many conferences with clinicians and rhinologists of large experience, that actual secondary infective heart lesions of an endocardial nature are rarely produced from sinus membrane disease, but that the toxemia produces more myocardial involvements than are commonly admitted in the literature.

To illustrate a typical example of secondary infection, the following report is given:

Report No. 3.—W. D., age 13. This patient was seen in consultation with an internist in an attempt to find a primary focus of infection. The heart was enlarged, the pulse rate high and the temperature running a septic course. There were no nose and throat complaints other than the general inflammation of the membrane of the throat and nose that one would expect with a convalescent influenza patient. The onset was two weeks previous to the time of examination, at which time there was an acute inflammation of the nasal membrane, with generalized headache. This condition subsided after a week, followed by a rise in temperature, marked generalized headache, high pulse and septic temperature, ranging from 99 to 105 degrees. Urinalysis was negative. Repeated blood cultures were negative. The blood picture was 83 per cent hemoglobin,

4,390,000 red blood cells, 12,750 to 13,500 white cells, of which 75 per cent were polymorphonuclear cells. There were no localizing symptoms referable to the sinuses but a rhinologic examination was requested. The right antrum was found to be completely filled with pus. The other sinuses were normal. Repeated irrigations were done, with immediate and constant improvement in the clinical picture. Here the picture is obscured by the possibility that the infection produced may have originated from the primary influenzal infection. Two blood cultures were sterile. However, the two interesting points are, first, the absolutely "silent" antrum, and, secondly, the striking improvement after the antrum infection was cleared. It is not necessary to assume that a clinically recognizable septicemia preceded this picture. Many infection syndromes occur without a stormy preceding illness. It does mean that a bacteremia must have preceded the condition and that bacteria were present in sufficient numbers or of such virulent character as to have survived the defensive forces.

In chronic pyogenic infections of the sinus mucosa the organisms may invade the submucosa and, with quiescent intervals, activate at times under climatic changes or under the stimulus of other infections. Any established infection in the nose is a constant menace to the lower respiratory tract. Anemia, cough, expectoration of mucopus sometimes streaked with blood, loss of appetite, wasting, and even nocturnal fever and sweats may occur in chronic purulent sinusitis. In some instances tuberculosis may be simulated. These septic sinuses are prone to cause recurrent pulmonary infections until in turn in neglected or resistant patients a subinfection in the lung may become a chronic focal infection.

Real secondary infection in advanced cases of sinusitis with marked tissue change is not observed with the frequency that it should theoretically be found, in view of the purulent character and extent of the pathologic changes. Manifestations of toxemia, such as loss of weight, strength, color, appetite, deep headache and lowered efficiency are common. It is probable that when a great absorption of bacteria occurs in adults it is due to some unusual combination of very virulent organisms. One organism may render the mucosa permeable to the other. The true explanation of any large bacterial leak is



usually that it is from some secondary sinus complication, such as an orbital cellulitis, or an osteomyelitis with a secondary dissemination. Most of the perforations, especially in frontal and ethmoid cells, with orbital cellulitis, abscess or meningitis occur in children while the sinus walls are thin. Sinuses are air-bearing cavities, and chronic states of the membrane are cured by ventilation and not by lavage, which is of little value in chronic sinusitis.

Many sinus procedures, even in the hands of recognized operators, would look mechanically defective and physiologically imperfect if they could but be better displayed. This is undoubtedly frequently due to lack of cooperation or "follow-through" spirit on the part of the patient. However, it seems safe to assume that prompt clearing up of sinus membrane disorders by efficient, thorough but rationally conservative treatment might do much to save damage to vitally important tissues and delay many of the degenerative changes of later life. Neglected sinus patients often look old beyond their years.

Sinus membrane has a great tendency to approach the normal state with capable surgical aid in restoring adequate ventilation when it does not contradict physiologic laws. If the mucoperiosteum is irreparably involved it should be entirely removed but such removal immediately exposes the surface to the danger of systemic absorption, so that it must be carefully protected until regeneration is well under way. The recent animal experiments of Knowlton and McGregor revealed that one month after the lining membrane of the antrum was removed epithelial regeneration was well established, that it was complete three months afterward, and that gland regeneration was well established, and that the mucoperiosteum looked almost normal five months afterward.

#### CONCLUSIONS.

Toxemia in chronic disease of sinus membrane is of great clinical significance and probably originates from absorption of small quantities of highly virulent toxins generated within the sinus cavities.

Whether the important amount of absorption occurs through the sinus membrane or direct from more highly absorptive

tissues which receive the toxic material from sinus drainage is a subject for further observation.

Actual secondary or subinfections are not seen resulting from acute and chronic disorders of sinus membrane with the frequency that theoretically should hold, in view of the purulent character and amount of pathologic change.

Clinical observation would seem to bear out the fact that secondary infections when found are usually blood borne.

Severe constitutional symptoms are seldom produced by uncomplicated disease of the sinus membrane.

Low toxemia continuing over long periods may have much to do with the progress of so-called degenerative changes of later life.

In severe acute sinusitis the systemic symptoms are frequently those resulting from the concurrent infection.

Physiologic or pathologic chemistry of the products of the diseased sinus membrane is an almost untouched field.

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## XVI.

### THE PREPARATIONS OF PAPERS ON MEDICAL SUBJECTS: HOW TO MAKE THEM INTEREST- ING AS WELL AS INSTRUCTIVE—THE EDITOR TALKS.\*

BY GEORGE L. RICHARDS, M. D.,

FALL RIVER, MASS.

At the outset I wish to define what I mean by the word interesting. An interesting paper on a medical subject is one that holds the hearer or the reader, as the case may be, until the end of the article. The word instructive needs no definition.

Since its organization the members of this society have contributed much of value to the science of medicine, as represented by our specialty, and have published their observations in medical journals as well as in several books. These contributions give a good moving picture of the progress in this department during the lifetime of the society and are a part of its permanent literature.

Our own published proceedings, now numbering 34 volumes, contain 15,000 pages, and of these we are rightly proud. We began with a modest paper-covered volume of 148 pages, and in 1925 there were 712 pages in the volume, which had long since outgrown its paper binding. At present our annual volume averages about 600 pages, and it requires some effort on the part of the editor to keep it within these limits.

For about sixteen years I have been your editor, and have prepared the papers from annual meetings and section meetings for publication. This has necessitated the reading of all manuscripts before publication and the twice reading of all proof as well as much correspondence.

As a result of these years of experience, I wish to present to you some conclusions to which I have come, and to offer

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\*Presented before the Annual Meeting of the American Laryngological, Rhinological and Otological Society at San Francisco, July, 1929.

some suggestions which if followed will make our papers and our Proceedings more interesting and just as instructive.

Medical literature at its best is not particularly exciting, whether presented by the human voice or through the medium of the printed page, and I am not the only person who has taken refuge in sleep during the presentation of a paper somewhat overlong and a bit prosy.

The amount of medical literature is now so vast that even the specialist in any one department finds it impossible to cope with it. He desires to keep up with medical progress, and when his journals and reviews arrive turns to those subjects which interest him. If the article is short he quickly reads it; if it is long he either turns to the end, hoping the author has summarized his conclusions, or lays it down on his desk, thinking he will read it another day when he has more leisure. By night it is probably covered by some other journal or piece of literature and the next day is covered still deeper.

In a few days another journal has arrived, and in the end the article never is read, even though the subject was an appealing one and by someone whose views would have commanded respect. Even the reprints which we mean to read are not read. Most of us are very busy and out of office hours like to do something else than read long articles and many case reports. Is not this in a measure the experience of all of you?

What is the remedy? To stop writing? Not at all, but each medical author should study the art of condensing and should write for his particular audience. In papers to be presented before this or similar societies one should assume that the audience have some familiarity with the subject, and too much historical matter or the relating of what all the other writers on the subject have said, starting with Hippocrates, might well be omitted and also all the anatomy except what is necessary to make clear the points of the paper.

At the beginning assume the audience have some knowledge of the subject, state clearly the point you are wanting to make and stop when you are through, after having briefly summarized.

Spell the same name the same way always and do not capitalize medicines, diseases and the like, unless they bear the

name of the originator. Rules as to capitals vary somewhat. It is my habit to follow the custom of the *Journal of the American Medical Association* as to these.

Before sending the manuscript to editor or printer read over carefully for grammar, capitals, spelling and clearness of statement.

Always read the manuscript carefully for typewriter errors. These naturally occur, but they have to be found by the editor, who cannot depend on their being found and corrected by the printer, especially when technical matter is being set up, the meaning of which may not be clear to him. If many corrections are necessary rewrite the page.

After the paper is written go over it and eliminate every unnecessary sentence and see that it reads smoothly. Then read it out loud to your secretary and see if you can hold her interest during the reading. This will be a fair test of how the audience will receive it.

If you are reading the paper get the attention of the last person in the audience; be sure he hears you and be sure you interest him. If you hold his attention you have captured all the rest on the way. Never have the word "louder" come to you but once.

Where possible, case reports should be summarized. It is not necessary to give the minutiae of each individual case unless that minutiae is essential to its proper understanding. Most of the family history can be omitted, whereas the previous history may be very important. Give only the essentials of the daily clinical history. Too many times a case report reads like a transcript of the hospital record. Where there is an autopsy report only what is relevant. Organs not mentioned may be assumed to be negative or it may be stated "other organs normal."

Do not use the word "she" or "he" and in the next sentence "the patient," alternating between these terms, as often occurs in many manuscripts. In a case history, once having given the sex and age, assume that the person about whom you are writing continues to be the patient.

I do not think it is good form to state that it is case No. 20,212 that you are reporting or that it was referred through the kindness of Dr. J. or H. When reporting a consultant's

opinion state the essential facts and not of necessity his complete statement. It is not always necessary to give temperature, pulse and respiration for each day of the report, nor, if a chart is reproduced, is it essential to give normal temperature for the convalescent period. The simple statement that from that time on the temperature was normal suffices.

#### BIBLIOGRAPHY.

I approach this subject with some fear that I may be misunderstood. Bibliographies are useful when they are distinctly referred to in the text and have some direct bearing on the subject, but when they are unduly long and have evidently been made by some energetic secretary who has culled from a medical library about all the references to be found on the subject, they appear to me to be somewhat superfluous.

This is especially the case when many of the references are foreign and are presented with the names of the journals badly spelled and with no uniformity in the abbreviations used.

One year we published a paper of  $6\frac{1}{2}$  pages which had 65 references in the bibliography. In this instance it was a report of four cases, and the references were all referred to in the text. Except, however, in the largest kind of a medical library it would have been impossible to verify these references. Fortunately they were correct as to spelling. That kind of a bibliography, even if long, is permissible and may be very useful to one looking up the subject.

On the other hand, in 1905 we published an article with 251 bibliographic references, none of which were specifically referred to in the text. These references may all have been valuable, but such a long list suggests padding and an attempt to show erudition and familiarity with the literature.

#### THE DISCUSSION.

Pleasant remarks on the paper and how we have enjoyed it and what a wonderful contribution it is to the subject are quite in order at the beginning or end of the discussor's remarks, but when the material is being set up for permanent reference in a scientific publication, be it medical journal or volume, they have no force and should be omitted.

The following is an example taken from the excellent *New England Journal of Medicine*, of May 23, 1929:

"I have enjoyed the papers of the evening very much indeed and feel that although much has been written on this subject there is always something we can carry away with us of value. I am sure the other members present feel as I do, that we have learned a great deal tonight."

Very pleasant remarks and no doubt justified but they add nothing to the value of the discussion, so why take the reader's time or put the *Journal* to the expense of reproducing them.

It has been my own practice, as perhaps you have noticed in reading your discussion as printed in our volume, to omit such paragraphs as well as remarks which are not pertinent to the subject of the paper. It is, of course, the stenographer's duty to take them down. I think it is the editor's duty, when he makes up his material for the printer, to omit them.

The proceedings of the Royal Society of Medicine of England, which are a model for conciseness, contain none of this complimentary material, but the discussion is always directly to the point. Perhaps one of the reasons for this is that no stenographer is, as a rule, present at the meetings, and the discussors are asked to write out their remarks immediately after making them and hand in to the secretary.

Where lantern slides are the main feature of a paper, whether of charts or illustrations, the paper should be prepared in two forms, one for reading and the other for printing. The paper intended for printing should be accompanied with clear drawings of such illustrations as are desired, with typewritten legends on the back and proper references to each one in the text and in proper order. All illustrations should be marked as to top and bottom, as in many cases the proper position is not clear to editor or printer unless so marked.

Bibliographic references should be referred to by number in the text.

Mrs. Mellish-Wilson of the Mayo Clinic has written an excellent little manual entitled "The Writing of Medical Papers," published by W. B. Saunders Co. Every contributor to medical literature should own and study this book.

## XVII.

### RETROPHARYNGEAL ABSCESS OF OTOGENIC ORIGIN.

By S. J. PEARLMAN, M. D.,

CHICAGO.

Retropharyngeal abscess, while not rare, is not a commonly seen lesion. Wishart<sup>1</sup> states that in 33,892 admissions to the Toronto Hospital for Sick Children in eight years, retropharyngeal abscess was found only 41 times, which would make the incidence only a little more than one-tenth of 1 per cent. Out of 15,462 admissions to the Sarah Morris Hospital for Children during the years 1923-1928, inclusive, there were 61 instances of retropharyngeal abscess, making an incidence of about .3 per cent of 1 per cent. Of these, 9, or 14.7 per cent, presented a concomitant otitis media, and 7 of these were on the same side as the abscess in the throat.

Although it is generally admitted that a retropharyngeal abscess may arise from the spreading of an otogenous infection through any one of a possible number of routes, yet the number of retropharyngeal abscesses which can be traced to such an otogenous source forms but a small proportion of the whole, at least so far as can be judged from the literature. In a series of cases cited by Wishart, about one-fifth showed the presence of an accompanying or preceding otitis media. It is probable, however, that otogenic infection may extend to the pharynx more frequently than is suspected, owing to the lack of association in the mind of most observers when a retropharyngeal abscess is noticed an otogenous source of infection is not suspected and hence is overlooked.

It is desirable, owing to the comparative infrequency with which otogenic retropharyngeal abscess is met, that every case should be published, and this is the reason for reporting the following personal case. A short survey of the literature is also given, as there is but little information on this subject in American textbooks or periodical literature.



## CASE REPORT.

E. B., a male white child, two years of age, was admitted to the Sarah Morris Hospital for Children on February 2, 1927, with the complaint of a head cold and cough two weeks in duration, irritability and fever for the past four or five days. A bilateral paracentesis to relieve an acute otitis media was done on the day of admission. The paracentesis had to be repeated on the right side a day or two later. Both ears then discharged freely for one month, during which time a febrile course prevailed. Local symptoms supervening, a simple antrotomy was performed on the left side. The discharge in the right ear by this time had become mucoid but was still profuse. Furthermore, about a week after the operation, a swelling appeared in the pharynx medial to and above the right tonsil. During all this time an irregular temperature was present, not definitely septic, however. A day or so later the pharyngeal swelling was opened with evacuation of considerable pus followed by prompt subsidence of the fever.

Guillemin,<sup>2</sup> in a thesis published at Nancy in 1913, collected 47 cases of otogenic pharyngeal abscess from the world's literature, including two unpublished cases of his own. The original report is not available here, but the facts are given by McKenzie,<sup>3</sup> who, in 1915, added 13 further cases, including one of his own. That this lesion is seldom or never seen by the general practitioner is testified to by the fact that Holmes<sup>4</sup> in 1907 sent out a questionnaire to 250 ear, nose and throat surgeons, and 147 replied that they had never seen a case of this kind. Ten or more surgeons reported that they had seen one or more such abscesses, altogether 16 cases among them. Chevalier Jackson reported that his records showed 12 cases of retropharyngeal abscess, with which an associated otitis media was observed. McKenzie Brown,<sup>5</sup> in 1919, in 5 cases of retropharyngeal abscess, found that one was preceded by otitis media. Wishart noted 7 such cases in 41 retropharyngeal abscesses and Guthrie<sup>6</sup> 4 in 20. Fulkerson<sup>7</sup> reported a case in 1916; Reynolds<sup>8</sup> one in 1917; Landry and Billard<sup>9</sup> one in 1927, and Cunningham<sup>10</sup> 5 cases in the same year, 4 of which were his own.

A few words regarding the anatomic disposition and relations of the retropharyngeal space will not be amiss in dis-

curring the routes followed by pus before collecting to form an abscess in this region.

A cross section at the level of the hard palate shows the so-called retropharyngeal space, bounded posteriorly by the *M. longus capitis* and *M. rectus capitis anterior*. Laterally the retropharyngeal space is continuous with the pharyngomaxillary (Zuckerkindl) or parapharyngeal (Corning) space. At the level of the tongue the pharyngomaxillary space is divided by the *M. styloglossus*, *M. stylopharyngeus* and the *M. stylohyoideus* into an anterior and posterior chamber, the latter in its hindmost part containing the internal carotid artery, the internal jugular vein and accompanying nerves. A little higher than this level the upper extension of the retropharyngeal space is limited by the fact that the subpharyngeal connective tissue binds the mucosa very firmly to the basilar process of the occipital bone. Downwards the retropharyngeal space merges into the retroesophageal space and continues to the thorax.

The pharyngomaxillary space is in close relationship to the parotid gland and its investing fascia; the parotid is on rare occasions peculiarly susceptible to ear infections, both in the middle ear and in the external auditory canal, and these inflammations spread easily to the pharyngomaxillary fossa and from here may invade the tissues of the retropharyngeal space.

Returning to the superior limits it is observed that the space between the atlas and occiput is also divided by the *M. rectus capitis lateralis* into an anterior and a posterior compartment—i. e., the subpetrous and suboccipital spaces, which are of particular importance in the genesis of retropharyngeal abscesses, spreading directly from infections of the middle ear and mastoid. In the suboccipital space lie the insertions of the deep posterior and lateral cervical muscles and the *canalis condyloideus*, important in the spreading of the same type of infection but through the medium of intracranial abscesses. Between the medial border of the *M. rectus capitis lateralis* and the occipital condyle there is only one layer of fascia: pus reaching the suboccipital region in any way and behind this muscle may easily break through this barrier, thus opening up a pathway to the pharynx.

Middle ear suppurations and their accompanying mastoid complications not infrequently invade the cranial cavity above and the digastric fossa below; very rarely does infection from the middle ear go forward to attack the pharynx because of the free drainage by way of the eustachian tube and the canal for the *M. tensor tympani*. Should the tube, however, be blocked by granulations, etc., suppuration may advance anteriorly and involve the pharyngeal wall about the tube.

McKenzie<sup>3</sup> says that there are four ways in which an otogenic abscess may arise:

1. By direct extension of a middle ear suppuration through the petrous portion of the temporal bone, which lies in close proximity to the pharynx.
2. Indirectly by the wandering of an extradural abscess of the middle cranial fossa to the tip of the petrous bone and thence by the foramen lacerum or otherwise to the extracranial, inferior surface of the petrous bone and thence to the pharynx.
3. By direct extension of infection from the pneumatic cells in the tympanomastoid osseous structure to the under surface of the occiput and thence to the pharynx by way of the suboccipital space.
4. Indirectly by the wandering of an extradural abscess of the posterior cranial fossa to the suboccipital region.

The first two of these McKenzie calls the subpetrous variety of otogenic retropharyngeal abscess and the last two the suboccipital variety.

Discussing these routes in order: First, as regards the subpetrous variety McKenzie remarks that the direct extension of a middle ear suppuration through the petrous bone, although the most natural route, is selected less frequently than the indirect route; in favor of the direct route from the middle ear through the petrous bone and so to the pharynx is the close anatomic relationship of the apex of the petrous to the latter. The near by bony eustachian tube may contain air cells in direct communication with those of the middle ear. Furthermore, the apex of the petrous may contain cellular extensions from the mastoid, the so-called supralabyrinthine cells, which pass between the cortex of the petrous and the upper limits of the labyrinth; so, too, sublabyrinthine cells may extend to the apex of the petrous from the tympanic floor

between the labyrinth capsule and the jugular bulb. The canal for the tensor tympani is thought by some to play a part because its position is such that pus may track down it so as to reach very near to the pharynx; some cases in the literature support this view. Guillemin,<sup>2</sup> however, criticises it on the ground that the muscle would be more likely to obstruct than to favor the propagation of any but a most virulent infection. If the canal were a likely route one would expect to see pharyngeal complications much oftener than one does.

Another possible direct route for pus to follow is by way of the carotid canal, and a considerable number of cases of this kind are recorded, in some of which there was a fatal hemorrhage through ulceration of this vessel.

Regarding the indirect route through the middle cranial fossa: Extradural abscesses of the middle cranial fossa arise usually in the roof of the tympanum, aditus or antrum, and to reach the apex of the petrous bone pus has to burrow downward, forward and inwards between the dura and the anterior surface of the pyramid. This pus must find its way out of the cranial cavity through the foramen lacerum, ovale or some other opening, such as a fistulous one. An extradural abscess of this kind may also occur as the result of disease of the petrous bone itself.

McKenzie, however, seems to feel that pharyngeal abscesses arising in this fashion are very rare; the close adherence of the dura to the bone of the petrous pyramid and its firm attachment to the margin of the foramen lacerum do not permit this route to be taken very often. However, should the pus reach the inferior surface of the petrous portion of the temporal bone it is in close relationship to the pharynx.

Regarding suboccipital otogenic retropharyngeal abscesses, McKenzie, as stated previously, divides the routes into direct and indirect ones, and states that extension of a suboccipital collection of pus to the pharynx is exception<sup>1</sup>. Suboccipital abscesses either remain limited to this region, pointing and discharging in the upper region of the neck behind the sternomastoid, or else they may gravitate to the lower cervical region.

He defines, furthermore, the suboccipital region as that part of the cervical region directly beneath the occiput and posterior to the styloid process and rectus capitis lateralis muscle;

it is medial to the inner aspect of the mastoid process, lateral to the margin of the foramen magnum and the occipital condyle and anterior to the superior curved line of the occiput. This definition brings the jugular foramen into the subpetrous region and so assumes that pus emerging from that opening will pass to the pharyngeal wall direct.

When pus from the middle ear and mastoid reaches the suboccipital region directly, it does so from the cells of the mastoid placed far back at the base of the skull. Some of these cells fall into a group studied by Cheatele and others, and called sinusodigastric cells, and may be so extensive as to invade the occipital bone. When fully developed the jugular foramen may be completely surrounded by these air spaces.

In the indirect route to the suboccipital space, through the medium of a posterior cranial fossa abscess, it has usually been assumed that the pus from such an abscess burrows down to the jugular foramen and finds its way through this opening to the pharyngeal region. Guillemin, however, opposes this view on the broad ground that the close adherence of the dura mater all around the foramen would prevent the passage of pus. He suggested as a more likely route that the pus may emerge through the occipitomastoid suture where it traverses the lateral wall of the jugular foramen, which would bring it directly into the suboccipital region.

Whether directly from a fistulous sinusodigastric cell or indirectly from the drainage of an extradural abscess, the extension of an infection from the suboccipital region may remain confined as a cervical cellulitis, opening externally, as happens in most cases; or exceptionally, gravitating toward the pharynx.

Cunningham<sup>10</sup> gives five routes in which an otogenic retropharyngeal abscess may occur:

1. By infection of a retropharyngeal lymph node.
2. By the direct extension forward of a middle ear supuration.
3. By direct extension from a necrotic area in the mastoid process.
4. By the exit of an extradural abscess from the cranial cavity.
5. By the secondary burrowing of a Bezold's abscess.

Beck,<sup>12</sup> in a comprehensive review of the literature of descending abscesses of otitic origin, states that Bezold and others claim that retropharyngeal abscess can only follow an acute otitis. However, experience has shown that it may complicate a chronic otitis. The pus may break through the tegmen and spread out in the middle or posterior fossa. From the middle fossa it may escape by way of the foramen ovale, rotundum or lacerum, and so downward. From the posterior fossa it may escape by way of the foramen occipitale magnum or jugulare, and so downward into the insertion of the deep neck muscles and forward, along the basilar part of the occipital bone, to the anterior surface of the vertebræ down to the level of the epiglottis, where the retropharyngeal space becomes very narrow. In this fashion the nerves in the foramen jugulare may at times be injured, with signs of paresis. Kessel<sup>17</sup> describes such a case with complete paralysis of the tongue, dyspnea, aphonia and very rapid pulse.

This indirect intracranial route is, of course, a rare source of origin for retropharyngeal abscesses. The direct source is more common. In this instance the pus escapes by way of the floor of the antrum or through the anterior bony auditory canal wall with possible involvement of the temporomandibular joint, or it may enter the posterior fossa through the posterior wall of the antrum and hence downward. The path along which the infection progresses may be in part preformed, as along vessel canals, or may be formed anew through massive bone destruction. The occipitomastoid suture may favor perforation, particularly if mastoid cells are present in this region. DeQuervain<sup>15</sup> mentions this possibility.

Perforation may take place through the anterior wall of the middle ear and advance toward the pharynx in the peritubal tissue. Haug<sup>10</sup> believes that the semicanalis tensoris tympani plays a rôle as a pathway for infection between the middle ear and the pharynx. In individual cases there may be a complete separation of the membranous from the bony tube. A direct breaking through of pus from the temporal bone toward the pharynx is possible, as Muck<sup>12</sup> and also Klug<sup>13</sup> as well as others have shown. Muck<sup>12</sup> describes a case in which, five weeks after the onset of an otitis media, a mastoiditis and an osteomyelitis in the spongy bone in the region

of the labyrinth and pyramid arose, causing an extradural abscess as well as a direct breaking through toward the pharynx and so producing a retropharyngeal abscess.

Gruber<sup>14</sup> has shown that breaking through from the ear into the temporomandibular joint may be followed by retropharyngeal abscess. Perisinus and peribulbar abscesses may spread themselves horizontally beneath the base of the skull and may invade the retropharyngeal space. Bezold's mastoiditis may be found combined with retropharyngeal abscess. Haug<sup>15</sup> described such a case. Furthermore, parotid abscesses arising from the middle ear, either by direct extension or by way of lymphatics, may invade the parapharyngeal space and thence the retropharyngeal space. Instances of otitis externa may be complicated by parotid abscess. As far as the diagnosis is concerned there are no great difficulties, because the relationship between the two is obvious. The diagnosis is not so easy when the ear manifestations have cleared up.

There is a certain amount of discussion in regard to the possibility of an otogenous retropharyngeal abscess occurring as the result of lymphatic infection. It has already been mentioned that there is a direct connection between the lymphatics of the ear and pharynx. The retropharyngeal lymph nodes are arranged in four groups in the retropharyngeal space, two on each side of the midline of the pharynx, the general arrangement being vertical.

McKenzie says that in infants, particularly the combination of septic infection of the retropharyngeal lymph nodes with suppuration of the ear, is not uncommon; even in adults unilateral swelling of the posterior pharyngeal wall (probably adenitis which has not come to abscess) is not unusual in suppuration of the middle ear. While it is more usual for such a septic adenitis to accompany a nasal or nasopharyngeal infection, yet it is occasionally transmitted from a middle ear infection. McKenzie, however, considers such a pharyngeal abscess to be metastatic and not direct.

Cunningham,<sup>10</sup> as stated, considers the lymphatics as one of the direct routes by which infection from the ear may pass to the pharynx; while the relation cannot be demonstrated, yet the coincidence of a suppurative otitis media and a retropharyngeal abscess, especially if on the same side, is sufficient

to render the relation extremely probable. One of Cunningham's personal cases was of this kind. Alagna's<sup>13</sup> case was also of this kind.

Retropharyngeal abscess as an extension of a Bezold's abscess is particularly stressed by Cunningham, who reports three personal cases of this type and also an unpublished case of a colleague, Dr. E. H. Saunders. As Cunningham shows, there is plenty of literature to support the view that a Bezold's abscess can extend to the pharynx.

Only a few words need be said in regard to symptomatology and treatment. In the earlier stages of the development of an otogenic as well as of any other type of retropharyngeal abscess, the main symptoms which suggest its presence are difficulty in swallowing, pain and stiffness on moving the jaw, localized edema and redness in the region posterior and medial to the tonsils and perhaps symptoms of septicemia. When the abscess is well advanced, if untreated, there should be some evidence of cervical cellulitis with later septicemia and pyemia unless the abscess resolves or points to the pharynx, when the local bulging, fluctuation, pain and other signs should be unmistakable. If the abscess does not point toward the pharynx the cellulitis may extend downward to the mediastinum, resulting in general sepsis and death. The most pronounced phenomena of abscess of the suboccipital region are those of cellulitis of the posterior triangle of the neck.

As far as treatment is concerned, in the case of a suboccipital collection of pus following a mastoiditis, opening of the mastoid and drainage will usually prevent the development of cervical cellulitis and subsequent pharyngeal abscess. This is the usual procedure today, and retropharyngeal abscess of this origin is hence rare. In the cases, however, of pus in the subpetrous region, operation is not so easy.

McKenzie recommends that an opening be made with a gouge in the anterior bony wall of the external auditory meatus so as to form a window close to the tympanum and deep to the temporomandibular articulation. This will lead to the subpetrous abscess.

If the pharyngeal wall is bulging it should be opened through the mouth. Unless widespread cellulitis is present and external opening is not necessary, no anesthetic should be employed,



and the posture of the patient should be such as to guard against pus entering the trachea and larynx. The ear should receive attention at the same time and the source and pathway of the infection be investigated. Cases of manifest retropharyngeal abscess so treated usually recover.

If a suboccipital abscess is still confined to this region, the operative treatment recommended by McKenzie is to detach the sternomastoid muscle, to remove the tip of the mastoid process and to work inward close to the digastric fossa and jugular process of the occiput so as to provide a free exit for pus in this region.

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180 NORTH MICHIGAN AVENUE.

## XVIII.

### TREATMENT OF MAXILLARY SINUSITIS.\*

BY JAMES J. KING, M. D.,

NEW YORK.

The maxillary sinus is the most frequently involved of all the nasal sinuses. The subject will be divided into the treatment of acute, traumatic, chronic and malignant maxillary sinusitis. My object in bringing this subject before this society today is not to present something new, but to elicit in the discussion something which may be beneficial to a considerable number of these patients in whom the treatment has not been entirely satisfactory. There are a certain number of them who continue to go on with their trouble in spite of continual and approved treatment. A certain number of the cases are of dental origin, while others arise from infection within the nose. All of them, therefore, are secondary infections.

#### GENERAL CONSIDERATIONS.

A warm, dry climate is beneficial to patients with maxillary sinusitis. To those patients who have a subacute or chronic sinusitis, a change to a dry, hot climate may be recommended. If this is not feasible, the hygienic and climatic conditions should be made as favorable as the circumstances permit. Fresh air and sunshine are always beneficial. Cold air, cold water or chilling of the body is not good. Patients are likely to do better if they do not swim while suffering from sinusitis.

Such local treatment as may reduce swelling in the nose and promote drainage is recommended.

Consideration should be given to the diet, as has been recommended by Dean and others. Rest is also important, especially in the acute condition. The use of the ultraviolet light I have found beneficial.

I have seen a few patients with a very acute purulent sinusitis which cleared up immediately after the administration

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\*Read at the Fifty-first Annual Congress of the American Laryngological Association, Atlantic City, N. J., May, 1929.

of ether by inhalation. This observation was made on patients who developed an acute mastoiditis and had to have mastoidectomies done. In several instances I have found maxillary sinusitis practically cured after the ether anesthesia.

Focal infections in the mouth, such as diseased teeth and infected tonsils, should be eradicated to facilitate the cure of all sinus conditions, whether acute or chronic. Some consideration should be given the proper time to remove tonsils in the acute cases. The subject will not be discussed here with too much detail or technic, as it is not necessary before this association.

#### TREATMENT OF ACUTE CONDITIONS.

In the treatment of acute conditions we irrigate the antrum at least once a day with a normal saline solution, at a temperature of 110° F. We use a Coakley trocar and cannula, and introduce it in the lower meatus of the nose underneath the inferior turbinate, about three-fourths of an inch from the anterior tip. The part is first anesthetized by spraying the nose with a 2 per cent solution of cocain to shrink the tissues. A pledget of cotton is then saturated with a 10 per cent solution of cocain or novocain and adrenalin, and is introduced between the inferior turbinate and the antral wall, and left in position for fifteen or twenty minutes. The length of time necessary to produce anesthesia varies very considerably with different patients. Novocain is used wherever possible instead of cocain. In some patients sufficient anesthesia is produced by merely applying the cotton-wound applicator once and withdrawing it immediately. In others it is necessary to leave it in position for a period of thirty minutes. Another method is to introduce a cotton-wound applicator with a cocain solution and gently rub the nasal wall at frequent intervals until the anesthesia is produced. It is sometimes desirable, where there is considerable congestion around the normal opening, to introduce a pledget of cotton saturated with a 2 per cent cocain and adrenalin solution in the middle meatus of the nose, near the normal opening. This will shrink down the tissues in and around the opening so that the contents of the antrum may be expelled without difficulty. It is absolutely necessary to allow plenty of time to effect complete anesthesia, otherwise

the procedure would be so painful that the patient would not stand for it. We are accustomed to having a number of patients in the office each day, undergoing antrum treatment, with these anesthetic solutions in the nose waiting for the anesthesia to take place.

It is difficult and unsatisfactory to treat acute antrum cases in clinics where the service is in session only every other day, as daily irrigations are necessary to effect a satisfactory cure.

We often see patients with acute sinus conditions where the whole upper respiratory tract becomes infected, including laryngitis, pharyngitis, tracheitis, tonsillitis and bronchitis. No progress can be made in the lower part of the tract so long as the upper respiratory tract is infected.

Our experience coincides with that of other observers, in that antrum disease originating within the nose presents pus of a somewhat different character from that which originates from an infected tooth. Where an infected tooth is the source of trouble we often find the antrum filled with a very foul-smelling, disintegrated pus, with an involvement of only one antrum, and this condition rapidly clears up under daily irrigations and appropriate dental care, such as the removal of the infected tooth or teeth. In the infected antra of nasal origin, usual as a sequel to common cold, the discharge is thick, yellow, not disintegrated and usually not foul smelling. In our experience, from 15 to 30 irrigations daily are sufficient to cure these acute cases.

It is not good practice to poke a hole into the antrum through the alveolar process for drainage. Epithelium grows in these openings and the mouth bacteria get there through the process of eating and at other times and keep up the infection. Where, in the extraction of a tooth an opening has been made into the antrum which persists, it is necessary to close up this mouth opening and irrigate through the nose as above described.

In acute cases, with the extraction of teeth, many get well promptly, but if the disease continues for any great length of time with a fistula from the mouth into the antrum persisting, it will travel from the antrum into the ethmoid and frontal.

Certain difficulties may arise during the treatment of acute antra. One of the most common is failure of the fluid to return from the antrum during irrigation. This may be due to a

blockage of the trocar. The point of the trocar may be imbedded in the thick mucosa and not within the antrum. The normal opening of the antrum may be occluded by swelling of the mucosa or by a polyp. The first two conditions mentioned should not occur, as they are errors of technic. Occlusion by means of a very thick mucosa or polypoid blocking of the normal opening of the antra must be given consideration. Unless the symptoms are urgent, I am accustomed to stop irrigations for a few days in the hope that the swelling may subside and allow the irrigations to be continued. If such symptoms as headache, pressure or sepsis from the antral contents arise, it is necessary to then make a large opening, as in the window operation for the treatment of chronic cases. If there is a markedly deflected septum blocking the drainage from the antra, a submucous resection should be considered, preferably after the acute process has subsided.

Occasionally we see acute conditions in teachers, clerks, patients from distant points and others who find it inconvenient or impossible to have daily treatments. In such, a window opening in the antrum for drainage and ventilation is justified by the circumstances.

In acute cases where there is high temperature, with profuse discharge of pus, we find rest in bed, good elimination of bowel and kidney, in addition to the daily irrigations, beneficial and necessary. As soon as the acute symptoms subside, we advocate the removal of focal infections, such as tonsils and dead and infected teeth.

#### TRAUMATIC INFECTIONS.

Traumatic infections of the antrum usually follow an injury, such as a blow on the cheek, a fall against some hard object or an automobile accident. There may be a fracture of the malar bone in these cases. The antrum is irrigated in this condition in a manner similar to that described above for the treatment of acute sinuses. In these cases the discharge will usually be bloody at first, and if not treated an infection may be superimposed and the discharge will become purulent or mucopurulent. Such cases, treated soon after the accident, usually clear up very promptly.

## CHRONIC MAXILLARY SINUSITIS.

In the treatment of chronic maxillary sinusitis I desire to make a plea for conservative measures. The treatment which will establish free drainage and ventilation of the sinus, with the least destruction of mucous membrane in the nose, is the one that will give most satisfaction to the patient.

In a large number of chronic antra very satisfactory results have been obtained by means of a window operation through the nasoantral wall to establish drainage. These have been so gratifying that I have had to do very few more radical or destructive operations on the antra.

The operation may be carried out either under local or general anesthesia. For a considerable number of these operations we have recently been employing synergistic anesthesia, consisting of the injection intramuscularly into the buttocks of magnesium sulphate, morphin and novocain, the administration of 10 grains of chloretone by mouth and the injection of some oil and ether into the rectum, as recommended by Gwathmey. Some cocain and adrenalin is applied to the nose to prevent hemorrhage and also to lessen sensation around the site of operation. These patients come to the operating table asleep or almost so, depending upon the amount of ether by rectum. We frequently have them come to the table able to cooperate and yet not sufficiently anesthetized to be unconscious. After the operation is over the oil and ether is siphoned from the rectum.

For local anesthesia, cocain is applied in the region of the anterior ethmoid nerves and the sphenopalatine ganglion. Cotton saturated with cocain and adrenalin is also placed under the inferior turbinate and in the inferior meatus.

The method of draining consists in fracturing the inferior turbinate, removing a small portion thereof (not more than the anterior third), exposing the antral wall, introducing a rasp or large trocar into the antrum and then enlarging the opening in all directions. This opening should be made low and posteriorly, rather than high and anteriorly, as low and posterior openings will cause less postoperative discomfort. A high anterior opening may involve the nasolacrimal canal. After the opening is made and the sinus exposed, the antrum

is packed with iodoform gauze for twelve to twenty-four hours and the patient kept in the hospital for a few days.

I have frequently been surprised to see what I thought was a very severe chronic maxillary sinusitis clear up immediately after such an operation, which established good drainage and ventilation. I have the patients come to the office a few times for treatment and inspection following the operation.

We have had great satisfaction from the insufflation of an iodine powder into the antrum after it has been opened, using a special powder blower with a cannula shaped like the Killian irrigator. This is introduced into the antrum and the powder blown in, while the patient exhales. Unless the patient is told to exhale, he may inhale some of this powder into the larynx, which will cause a disagreeable coughing fit.

After the performance of such an operation, I have examined these patients with an antrumscope, and it has almost seemed as if it were possible, from day to day, to see the swelling and edema of the mucosa subside, due to the ventilation and drainage.

The mucosa of the antrum has great ability to repair, and the sinus with ciliated epithelium is better able to perform the normal function than one lined with stratified squamous cells.

The window is brought forward and down to the floor of the nose. If this opening is sufficiently forward, there is little danger of its closing, especially if a flap of periosteum and mucous membrane is saved and laid over the raw edge. The edges are smoothed with a rasp. No curetting should be done in the antral cavity.

The disadvantages of the radical operation are the infra-orbital neuritis, devitalization of teeth, anesthesia of the cheek, stenosis of the tear duct, osteomyelitis of the superior maxillary.

Where it is deemed necessary to explore the antrum, this should be done through the canine fossa.

There is a small percentage of patients with polypoid degeneration in the antrum or necrosis of the floor or antral wall, tumors, new growths or cysts, requiring more radical treat-

ment. In a recurrence of polypi of the nose, the frontal, ethmoid and maxillary sinuses must be explored. Hirsch has demonstrated that, when polypi recur, the antrum disease has probably been overlooked. Zuckerkandl has shown that polypi may be in the middle meatus with the ethmoid clear. Hirsch accordingly claims that polypi occur in the middle meatus and external surface of the ethmoid cells because the path of the disease outward from the antrum lies past the ethmoid. He also says that 70 per cent of the origins of polypi are overlooked, because 70 per cent rise from the antrum. He further states that polypi may arise from frontal and ethmoid disease, but his main contention is that the majority are due to antral disease.

When an exploratory operation is necessary, it is best to go through the alveolar process. In this small percentage of patients who need more radical treatment we recommend the Caldwell-Luc operation or a modification of it. I again emphasize that this operation should be done as infrequently as possible. All conservative methods should be given a fair trial before this is resorted to, except in patients with bone necrosis or persistent and recurring polypi, new growths, etc. Many cases simulating polypi, as shown by the X-ray, will recover when the window operation is done. I am thoroughly convinced that the less operative interference there is the better off the patient will be. When, after exploring the antrum, the membrane is found to be polypoid or necrotic, it should be removed, otherwise not.

#### MALIGNANT DISEASE OF THE ANTRUM.

In the treatment of malignant disease of the antrum it is necessary to remove the growth as completely as possible. When this has been done the application of radium should be considered. I am not certain that the application of radium will effect a better result with an operation than the operation alone. The application of radium in other parts of the body has been disappointing. It has been recommended that 10 mg. of radium be applied within the antrum for twenty-four hours, using two tubes in order to get a wider dissemination. The prognosis of malignancy of the antrum is better than that of cancer in other regions.



## CONCLUSIONS.

1. Daily irrigations with normal saline solution at 110° F., with an appropriate general treatment, is generally sufficient to cure the acute case in from fifteen to thirty days.

2. A window operation, giving ventilation and drainage with the least possible destruction of tissue, has been highly satisfactory in the treatment of nearly all chronic cases with the exception of those with polypi, bony necrosis or new growths.

3. In the small percentage of chronic cases which do not recover with the window operation, the Caldwell-Luc operation or a slight modification thereof is recommended.

4. Extensive removal of the growth is necessary in malignancy.

5. In spite of continual and approved treatment, there are still a few patients in each class whose progress is unsatisfactory.

30 EAST FORTIETH STREET.

## XIX.

### POSTDIPHTHERITIC PARALYSIS.

BY ALLEN B. POTTER, M. D.,\*

ST. LOUIS.

Postdiphtheritic paralysis has been termed by some authors as convalescence paralysis because of its usual late appearance, most often after all local manifestations of the disease have subsided and, so, often after the patient has been discharged by the physician.

Statistics on reported cases would show that there has been a noticeable increase in postdiphtheritic paralysis. This is accounted for by the fact that with the more judicial and extensive use of antitoxin more patients have lived through the toxic stage of the disease and have been permitted to experience the period of convalescence at which time the postdiphtheritic paralysis is noticeable or manifest.

Postdiphtheritic paralysis has been reported as occurring in about 10 to 20 per cent of all cases, and I believe this is the percentage accepted by most authors.

#### PATHOGENESIS.

This seems to be undecided. A number of writers have presented different opinions, while others are noncommittal. Walshe,<sup>1</sup> in an extensive discussion, wrote as follows:

"Mendel attributed the palatal paralysis to the fact that the nerves of this structure were bathed in toxin from the neighboring infective focus; but Bismarck does not accept this, on the ground that the other cranial nerve palsies are not explainable on this basis.

"Babonneix, 1904, put forward the view that the process was of the nature of an ascending neuritis from infective focus analogous to the nervous involvement in tetanus and rabies."

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The different views are: Lymphogenous; hematogenous; ascension along the nerve trunk.

Palatal paralysis develops as early as the sixth or as late as the thirty-fifth day, and persists from two to twelve weeks.

#### MANIFESTATIONS AND TYPES NOTICED.

Walshe,<sup>2</sup> in another article, reported an extensive observation, and shows that "in extrafaucial diphtheria the palatal paralysis, so characteristic an initial symptom in faucial diphtheria, does not occur, but that the fully developed syndrome consists of three essential elements, namely:

1. An initial local paralysis or paresis related to the sight of the infective focus.
2. A specific element consisting of the paralysis of accommodation.
3. A generalized element consisting of a polyneuritis."

And he goes on to say that "the accommodation paralysis may, therefore, be regarded as the manifestation of a selective affinity of the toxin for certain elements of the nervous system, while the polyneuritis is the expression of the general toxemia of that system."

Mixsell<sup>3</sup> quotes Osler as stating that "postdiphtheritic paralysis may have no relation to previous treatment, and the type, neither mild nor severe, determines its occurrence." "But," says Dr. Mixsell, "in our opinion there is a distinct relationship of the initial angina to the frequency and severity of the paralysis. In other words, the more severe the diphtheritic attack the more frequent and severe are the various paralyses liable to be; and in no type does this follow more closely than in the diaphragmatic type. . . . The early administration of antitoxin by the intravenous route determines the liability of the paralysis."

He continues: "Diaphragmatic paralysis is due primarily to degeneration of the phrenic nerve, which supplies filaments to the diaphragm, pericardium and pleura. The action is the result of a toxic peripheral neuritis, a parenchymatous degeneration of the nerves, and is not of central origin. The direct application of toxin to the nervous system causes a degeneration and an inflammatory action starting at the point of application and spreading from that point on. The toxin first at-

tacks the myelin sheath, then the nerve and finally the nerve center. This slow course would explain the late onset. The degeneration affects the motor and sensory fibers alike. (Ehrlich's theory that paralysis is caused by *toxon*, while the general poisoning is caused by *toxin* has been confirmed by later observers.) (A *toxon* is a toxin in which the toxophore group has diminished the toxicity.) Diaphragmatic paralysis never occurs but in conjunction with multiple paralysis."

Most of the reviewed cases of postdiphtheritic paralysis presented essentially similar findings with slight variation in onset, intensity, extensity and duration of the paralysis.

The structures involved may be briefly listed in order of frequency and intensity: Palate, oculomotor, ciliary muscles, eye (extrinsic), pharynx, face, levator palpebra superioris, trunk, larynx, respiratory muscles.

The sterno-cleido-mastoid involvement has been mentioned in a number of reports, this involvement being manifest by either weakness or tenderness to pressure of the muscle.

On the other hand, the only muscular involvement present may be a weakness of the leg or arm muscles.

Cutaneous sensation may be noted.

Parasthesia may be present and associated with muscle weakness.

#### REFLEXES.

Nerve involvement may be manifest by diminution of reflexes—plantar and patellar; also KK.

Ankle jerk in a certain percentage of cases was first to go and last to return.

#### CASE REPORTS.

Report No. 1. By Dr. Thorp<sup>4</sup>: "Nurse in attendance on case of diphtheria developed diphtheria. Was given 10,000 units of antitoxin, later 6,000 units. Disease ran an uncomplicated course and the patient reported for duty.

"Six months later she developed a well marked paresis of the muscles of the right cheek and eyelid, some difficulty in speaking, some loss of taste and complained of diplopia. After four weeks the paralysis disappeared."

Report No. 2.—This patient manifested six definite paralyses, but presented nothing of special interest except that

there was a well developed paralysis of the tongue, unilateral. This was an exceptional case in this respect, and reference to it was the only one made to that type of paralysis in the review.

Report No. 3.—The only case of permanent paralysis found in the review was referred to by Dr. Lombardo,<sup>5</sup> who, mentioning the original article by Duane (*Archives of Ophth.*, XXVI, 1927, page 317), reports that paresis of both external recti and secondary contraction of the internal recti were noted.

Reference was also made to an article (Tr. of the Kingdom, London, 1904, XXIV, page 238), in which was reported a case of paralysis of the inferior rectus muscle following diphtheria two years previous.

Report No. 4.—Dr. Scheffield,<sup>6</sup> in a report, writes as follows: "In connection with paralysis of the cranial nerves, it may be here noted that several cases are on record in which other cranial nerves were affected and that organic lesions (thrombus, embolus, hemorrhage) were found in other parts of the brain which led to permanent paralysis."

No exact reference was made in this report to the cases mentioned.

Boostein<sup>7</sup> draws the following conclusions from cases observed: "In cases in which the extremities are involved there is first a weakness followed by paresthesia, then pain, then follow disturbances of sensation and of coordination. Westphal's sign is always present (weakness or loss of tendon reflex). Rhomberg's is frequently noted."

Extract of a treatise by Dr. Edwin Hemphill Place:<sup>8</sup>

"Diphtheritic paralysis is due to the toxic degeneration of the peripheral nerves, the cell escaping. The paralysis is transient and without residuals, is incomplete in practically every involved nerve and is likely to be of widespread distribution. Both motor and sensory fibers are involved, but the sensory are much less susceptible, so that while motor paralysis is present and often alone, sensory paralysis is present and usually appears a little later.

"Early administration of antitoxin in large doses will decrease the likelihood of paralysis.

"Paralysis never results from antitoxin.

"A striking progression of paralysis in faucial diphtheria often occurs, appearing first in the throat, eyes, face, larynx, neck, arms, trunk, and legs, and clearing in the same order.

"Recurrent paralysis has been noticed in the palate (explained perhaps by local effect first, then by nerve involvement).

"Frequency of the paralysis varies with the severity of the case, the efficiency of the antitoxin treatment and care used to detect the condition.

"Sensory paralysis may show loss of tactile sense or pain, or both. Pain sense, especially, may be lost while tactile remains.

"Patients may have a typical tabetic picture with ataxia, Romberg's sign, and loss of KK. Paralysis of the smooth muscles apparently never appears. The sympathetic system involvement has not been demonstrated.

"Hemiplegia and monoplegia of central origin may occur during diphtheria, but they are rare and are due to hemorrhage or thrombus and not directly to the toxin. They, of course, may persist, in contrast to the diphtheritic paralysis.

"McCulloch has recently reported excellent electrocardiograms of diphtheritic cases, showing various degrees of degeneration in the bundle of His, from slowing of conduction to auricula-ventricular block."

From the casual observation of a few cases, one may infer that postdiphtheritic paralysis may be manifest in a haphazard manner; that the onset may be early or late, mild or severe, decidedly localized or general, with no relation to the extensiveness, intensity or location of the initial lesion. But after reviewing the available articles which have appeared during the past forty years one is more inclined to believe that there is more or less constancy in the manifestations.

In the faucial type palatal paralysis will be seen fairly early and in almost a hundred per cent of cases. The involvement of the third nerve and the general neuritis with its muscle weakness, alteration of the superficial and deep reflexes, changes in cutaneous sensation and even to actual muscle paralysis bear a rather constant relation to each other and to the intensity of the local lesion.

One must not overlook virulence of the organism and susceptibility of the individual, also the time and method of administration of antitoxin, as these are factors which must be considered during the observation of the cases.

In the extrafaucial type the palatal paralysis is never present. The third nerve paralysis is noted in only a small per cent of cases. General neuritis is fairly common and not unlike that which follows the faucial type, even to the marked interference of sensation, reflexes and varying degrees of muscular paralysis, again depending on the location of the lesion and the method of administration of treatment.

In conclusion, I feel it would be interesting to note the observations of a large number of cases made by Dr. F. J. Woollacott and reported in *Lancet*, August 26, 1929:

Year	All Complicated Cases	Antitoxin Cases Treated	All Paralysis Cases	Percentage of Paralysis	Deaths from Paralysis	Percent of Death from Paralysis	Total Mortality Percentage
1892-93-94	1523		174	11.4	19	1.24	33.8
1895	641	276	103	16.0	9	1.4	25.5
1896	633	475	136	21.5	9	1.42	17.3
1897	1060	980	161	15.1	6	0.56	17.2
1898	1301	1280	164	12.6	7	.53	15.9

It is also well to note that 1895 was the first year in which the antitoxin treatment was extensively used.

#### SUMMARY.

1. From the cases reviewed it appears to be generally conceded that the postdiphtheritic paralysis bears a direct relation to the severity of the attack.

2. Early and sufficient administration of antitoxin is an important feature in decreasing the liability of postdiphtheritic paralysis.

3. The motor nerve is the most often affected; the sensory least, of which pain and tactile are affected more often than temperature sense. Sympathetic is never involved.

4. That there is ever any residual effect is doubtful, since there were found only two rather indefinite case reports.

5. Paralysis of the eighth nerve was not mentioned in any of the reports reviewed.

#### CONCLUSIONS.

By carefully noting the structures involved and the nerve supply of each, it may be observed that the likelihood of the involvement of a nerve has no relation to its distribution, its shortness of course, its central origin, but does seem to have a definite relation to the percentage of motor fibers making up the nerve. In other words, a nerve purely motor or containing only a small amount of sensory fibers, is more apt to be involved than one which contains a smaller amount of motor fibers and more sensory. The first, second and eighth nerves, being the only cranial nerves escaping, are apparently immune.

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## XX.

### THE RELIEF OF NASAL OBSTRUCTION IN CHILDREN DUE TO DEVIATION OF THE SEPTUM.

BY WILLIAM WESLEY CARTER, M. D.,  
NEW YORK.

This subject has received considerable attention during the past decade from some of our ablest rhinologists, among whom there is a difference of opinion as to the method that should be employed in giving relief and the extent of operative interference permissible at an early age when nasal development is incomplete. No one questions the importance of giving a free nasal breathing space to the child. The deterring factor is the thought that an extensive operation on the nasal septum at an early age may interfere with the normal development of the nose, the result later in life being a deformity of this important organ, or at least a disproportion between its shape and size and its facial environment. Upon this important point there seems at present to be a decided diversity of opinion.

While I do not question for one moment the sincerity of those who have stated that the extensive removal of the septum in the very young is not followed by developmental defects, I feel that in the light of my own experience, I am warranted in making the suggestion that they have not had their cases under observation long enough. Apparently some of the clinicians who are interested in this subject have confounded the immediate effect of removal of the mechanical support of the septum with the more remote results due to interference with the developmental forces. The latter cannot be determined at once; frequently they do not become manifest for several months or even years after the operation; therefore, it is unsafe to express an opinion in these cases until a sufficient time has elapsed. I repeat with emphasis and with a knowledge gained by personal, clinical experience, that those who state that the nose of a child is not injured by the sub-

mucous operation have either not removed enough of the septum to interfere with development, which is a legitimate and laudable procedure, or they have expressed their opinions prematurely.

The construction of the nasal eminence is, to a large extent, a mechanical problem. It may be compared to the raising of a tent, the skin and subcutaneous tissues corresponding to the canvas. The elevating forces are transmitted through the septum and the nasal processes of the superior maxillæ. If the force exerted through any one or all of these three avenues has been interfered with in early life, the result will be a deviation from the normal, if not a frank deformity. This is the logical and practical outcome of the operation of definite mechanical laws and therefore there is no uncertainty about the results.

The total absence of these forces is well exemplified in many cases of congenital syphilis, where the nasal processes of the superior maxillæ as well as the septum, owing to a rarefying osteitis in these bones, have not exerted their developmental function. Here we have a broad, flat nose with practically no bridge: an exaggerated type of the infantile nose.

The clinical evidence that the septum is a prominent factor in the development of the flattened nose of the infant into the more prominent organ of the adult is so overwhelming that I believe no one could successfully defend a contrary position.

Now what shall we do with a child having a pronounced nasal obstruction, due to deflection of the septum? It is important to know how long the obstruction has lasted. If it is due to recent traumatism, it may be possible to realign the segments by means of my nasal moulding forceps and then hold them in proper position by introducing one of my gold wire splints into each nasal cavity. In older children the bridge splint may be used to advantage. This instrument, essentially an adjustable suspension bridge which I devised many years ago, and which I have used with great satisfaction, applies force along the lines that tend to reconstruct the nose, but owing to the fact that it must remain in position at least ten days it is better adapted to recent fractures in adults who may be relied upon not to disturb its position.

The deflected septum of long standing is a menace that calls for ingenuity, manual dexterity and a clear understanding of the dynamics of nasal development.

In operating, the prime consideration must be the preservation of the basic nasal developmental factors. The septum is by far the most important and must be preserved to the greatest extent consistent with the relief of the obstruction. (1) It is important that the anterior edge of the septum should be preserved, as this is the prop that supports the tip of the nose. (2) It is essential that the upper edge of the cartilaginous septum should not be dislodged from its position between the lateral cartilages, for here it constitutes the keystone of the cartilaginous bridge of the nose.

It is impossible to restore the deflected septum without some operative measure; the resiliency of the cartilage and the resistance of the nasal bridge render valueless simple packing or splints introduced on the obstructed side with the idea of gradually straightening the septum. I have tried out the idea as an experiment on several patients where consent for operation could not be secured, but I had no success. We may be sure that operative measures alone will afford permanent relief.

In dealing with these cases the chief obligations that we have upon us are to safeguard the strategic points of development and to save as much of the septum as possible. It is necessary, however, that the resiliency of the latter should be destroyed by incisions. These should be so directed that the segments can be lined up in a vertical position.

During the past six years I have used the following procedure upon a number of patients, ranging in age from five to fourteen, and so far I have noted, in those that I have been able to follow up, no tendency to the development of nasal deformity.

Operation.—Unless the child is exceptionally amenable, I prefer the use of ether anesthesia, the patient having received by hypo 1/200 gr. of atropin one hour before operation.

It must be remembered that the mucoperichondrium is thicker and more closely adherent to the cartilage in young patients than in adults, and therefore we must be more careful in elevating it and be certain that we are separating it from the

cartilage and not splitting it; this mistake can easily be made, as it is often necessary to use a sharp elevator in liberating the mucoperichondrium, especially along the lines of a fracture.

The usual vertical submucous incision is made reaching from the vestibule well down to the floor of the nose on the side of the deflection. After elevating the mucous membrane on this side, I introduce my small submucous knife into the sac and make two parallel incisions from behind forward on either side of the apex of the deflection. These incisions are made through the cartilage, but not through the perichondrium, on the concave side. The very narrow strip of cartilage lying between the above incisions and constituting the apex of the deflection is then removed. The object in removing this strip of cartilage is to permit the edges of the upper and lower segments of cartilage to fall together, superimposed without overlapping when they are lined up vertically.

An incision is then made through the upper segment, parallel with the dorsum of the nose and extending the entire length of the deflection. A corresponding incision is made below, parallel with the floor of the nose, if the vomer is involved in the deflection. The intranasal chisel must be used in mobilizing this segment; if, however, the vomer is not involved, the line of incision in the cartilage is carried along the anterior edge of the vomer.

It is absolutely necessary that the resiliency of the cartilage shall have been destroyed by these incisions, otherwise the operation will be a failure.

A summary of what we have accomplished so far is as follows: The septum consists of an upper and lower ribbonlike segment of cartilage and also bone if the vomer and the vertical plate of the ethmoid are involved. Between these two segments there is a slight interval corresponding to the apex of the deflection. Both of the segments remain attached to the perichondrium on the concave side, which has not been perforated, the mucous membrane on the convex side only having been elevated.

By means of my special moulding forceps, one blade of which is introduced on either side of the septum, we make sure that all resiliency has been destroyed; at the same time

we line up the septum in its normal, vertical position. I then introduce one of my gold wire splints into each nasal cavity. These splints are moulded at the time to suit the case. They are made from wire such as is used by dentists in straightening the teeth. The advantages of these splints over vaselin gauze packing is that they are resilient and hold the mobilized segments of the septum in proper position. They enable the patient to breathe through his nose, and they facilitate the cleansing of the nasal passages. The splints should remain in the nose for at least ten days, the nose being cleansed each day with an alkalin solution.

Immobilization of the nose is essential; this is effected by using one of my tin lined copper splints, which fits accurately over the nose.

In conclusion, I will say that results secured by this method are satisfactory in establishing a free breathing space, and so far, I have noted no deformity attributable to interference with the developmental force normally exerted by the septum.

2 EAST 54TH STREET.

## XXI.

### LABORATORY AIDS IN EAR, NOSE AND THROAT CONDITIONS.

BY CHARLES E. CONNOR, M. D.,

ST. PAUL.

Blood studies are capable of yielding a wealth of information in diseases of the ear, nose and throat. The diminution in red cells and hemoglobin in sepsis, especially of hemolytic streptococcus origin, has long been known and can be made use of in gauging the status and progress of severe septic aural processes; the loss of red cells may vary from 200,000 to 1,000,000 per week and they may drop to 100,000 in a few days.<sup>1</sup> The loss of hemoglobin may parallel or outstrip that of red cells. Kopetzky<sup>2</sup> finds such studies of great value, especially in cases of hemorrhagic mastoiditis, due to the streptococcus hemolyticus. The progressive loss of red cells and diminution of hemoglobin in a case of otitis media with septic temperature suggests such a lesion and may offer early operative indications. A favorable postoperative course is attended by gradual increase in red cells and hemoglobin; should progressive loss continue extension into the lateral sinus is to be suspected and a transfusion of whole blood given. If the reduction still continues the aural infection should be eliminated as far as possible from the general blood stream by suitable surgical measures.

Infection changes the white cell picture markedly. There is usually an absolute and often relative increase in the neutrophils; eosinophils and basophils diminish or disappear and lymphocytes are much diminished. During convalescence the neutrophils gradually return to normal, the eosinophils reappear and the lymphocytes increase until they may even exceed the normal number, the postinfectious lymphocytosis. The monocytes also show an increase during convalescence.

The total white count and percentage of neutrophils has long been accepted as a guide to the reaction of the patient and the severity of the infection. This is especially true in acute sepsis,

in which the leukocytosis may vary from a normal of 8,000 to 45,000 or 50,000 or more. Wicart<sup>3</sup> found the following values in aural sepsis:

	White Cells	% of Neutrophils
Acute suppurative otitis media	6,000 to 10,000	70 per cent
Acute suppurative mastoiditis	10,000 to 12,000	72 per cent
Thrombophlebitis	20,000 to 25,000	92-95 per cent
Circumscribed meningitis	20,000 to 25,000	94 per cent
Diffuse meningitis	35,000 to 60,000	92-95 per cent
Cerebral abscess with encapsulation	20,000	88-95 per cent

Relatively few acute infections, such as malaria, typhoid, influenza, measles and mumps, are attended by leukopenia. Leukocytosis depends on the resistance of the patient and the severity of the infection. When resistance is good and infection severe leukocytosis may be early and pronounced; if these two factors are less marked but proportionate the reaction is less sharp, comes later and disappears sooner. If resistance is poor or infection overwhelming an actual leukopenia may develop, well illustrated by agranulocytic angina. Successful surgical intervention in sepsis should be attended by a decrease in the total white count and percentage of neutrophils; thus Schmidt<sup>4</sup> found a marked diminution in the leukocytosis of peritonsillar abscess, which often ranges from 15,000 to 23,000, after incision, while interference in pharyngeal phlegmon caused no change. He considers the white count of more diagnostic significance in such cases than the temperature curve. Should leukocytosis and neutrophilia persist after surgical interference continued sepsis should be suspected; should these factors increase after having dropped, the onset of a complication should be thought of.

Study of the neutrophil nucleus as a guide to the progress of infection was proposed by Arneth<sup>5</sup> in 1904, when he showed that during infection cells with immature nuclei appear in the blood and disappear with convalescence. He at first divided the nuclear forms into five classes as follows:

1. Vesicular, round or slightly indented, or simple but deeply indented nuclei, 5 per cent.
2. Nuclei in two segments, 35 per cent.
3. Nuclei in three segments, 41 per cent.

4. Nuclei in four segments, 17 per cent.
5. Nuclei in five or more segments, 2 per cent.

A further subdivision of the neutrophil into 20 groups was made and a classification of lymphocytes and monocytes so extensive that the total number of divisions was eighty-one. There has not been wide agreement with Arneth's theory that the age of the neutrophil is exactly indicated by the complexity of the nucleus. The greatest difficulty with his classification was the practical one of recognizing minor variations in nuclear form when obscured by pyknosis or degeneration. The method was cumbersome and was not used extensively until Schilling<sup>6</sup> simplified it by using only four divisions. the myelocyte, normally seen only in bone marrow, the young metamyelocyte (W cell, jugendliche), rarely seen in the peripheral blood, the old metamyelocyte (T cell, staff cell, band cell or stabkernige), and the adult segmented neutrophil, which comprises about 63 per cent of the total count. This classification has made the procedure much simpler so that it may be used with but little loss of time.

Nuclear metamorphosis is indicative of the changes going on in the bone marrow. White cells come from three sources. Granulocytes (neutrophils, eosinophils and basophils) come from the bone marrow, lymphocytes from lymph glands and lymph marrow, and monocytes (large mononuclears and transitionals) from the reticuloendothelial system. Infection or toxemia may influence the bone marrow, the effect ranging from slight or strong stimulation to injury or destruction. These changes are reflected in the type of neutrophil. Slight irritation produces an increased output of normal neutrophils; with stronger stimulation immature forms make their appearance until in extreme cases the mother cell, the neutrophilic myelocyte, appears.

In making the count the young forms are put on the left side of the page and the mature forms on the right. In infection the percentage of young cells increases, hence the "shift to the left" or "linksverschiebung," and with convalescence the shift to the right or "rechtsverschiebung." Arneth counted 100 neutrophils, his figures being, therefore, percentages of the neutrophils, while Schilling counted each of his four classes while doing an ordinary differential, his results giving him per-



centages of the total leucocyte count. The result is known as the Arneth or Schilling Hemogram.

White cells play different rôles in inflammation. The neutrophil is a very sensitive indicator of the presence and severity of infection and degree of resistance and has long been made use of in the estimation of leukocytosis and neutrophilia. Return to normal indicates subsidence of infection but usually takes place later than the corresponding change in eosinophils, lymphocytes and monocytes. A drop in the absolute number of leukocytes with persistence of neutrophilia and shifting to the left is a bad sign inasmuch as the decreased number of cells indicates lessened bone marrow reaction while the neutrophilia and shifting to the left show that the irritative lesion is still present.

Eosinophiles respond to infection more exactly and quickly than do neutrophils; with its onset they diminish or disappear, and with its subsidence they reappear, and may be present in unusually large numbers, constituting the postinfectious eosinophilia. One must, of course, rule out helminthiasis, vagotonia and eczematous conditions which of themselves produce eosinophilia.

The rôle of the basophil is not well understood.

Lymphocytes react quite differently from neutrophils. In the presence of infection the total number may vary from 10 to 18 per cent instead of the normal 23 to 25. A lymphopenia, therefore, usually accompanies a neutrophilia and if it becomes marked and persists in the presence of acute infection, constitutes a bad prognostic sign. With the subsidence of infection, the percentage of lymphocytes increases, and in cases in which the disease is being held in check, convalescence or chronicity, increases to normal or above normal and constitutes the postinfectious lymphocytosis, a condition which is especially noted in children.

The monocyte is usually increased at the time of crisis, dropping slowly during convalescence.

Schilling has divided the leukocytic response to infection into three phases. The first he calls the neutrophilic phase of resistance; it is characterized by neutrophilia, shifting to the left, reduction in the number of lymphocytes and monocytes, and disappearance of the eosinophils. The second stage is the

monocyte defense reaction, characterized by increase of monocytes, return of eosinophils, reduction of neutrophils and persisting shift to the left. The third stage is the lymphocyte phase of recovery; it is characterized by shifting back to normal, by increase in the number of lymphocytes and eosinophils and by diminution in the number of neutrophils and monocytes.

The hemogram is used to determine the severity and progress of infection. Some writers find the so called staff count (referring to the immature or unsegmented "staff cell," "stabkernige" or "band form") of more value than the ordinary white count and differential, and frequently follow the case by repeated differentials with only an occasional leukocyte count. Kopetzky<sup>2</sup> finds it of inestimable value, especially in borderline cases, and recommends its more general use. He feels that 15 per cent of staff cells means continued infection, that 12 per cent means operation, and that any figure below 12 per cent permits further observation. Gale<sup>7</sup> reports the result of 250 counts made in suppurative ear lesions and does not find the hemogram a reliable guide to operative interference.

Hesse<sup>8</sup> found the total white count markedly increased in acute suppurative otitis media and mastoiditis, the higher counts meaning probable bone involvement; shifting to the left may or may not occur in acute, uncomplicated mastoiditis. He was unable by this means to separate cases of acute, suppurative otitis media with mastoiditis from those without mastoiditis. The most valuable aid of the Schilling hemogram is in the recognition of major complications, but a differentiation of complications is not possible.

Heidemann<sup>9</sup> feels that one must know quite definitely the limits of value of the blood picture in otogenous disease. One cannot wholly differentiate a simple otitis media from mastoiditis, even though something may be learned of the severity of the infection; occasionally operative indication is given. The fact that the blood picture may be normal in severe mastoiditis must be borne in mind. Chronic suppurative otitis media makes no change in the blood, although acute exacerbations show much the same picture as an ordinary acute infection. There are no changes in otosclerosis.

Heidemann draws the following conclusions: Occasionally the blood picture may offer operative indications in borderline cases but it is not of great importance in the average case of mastoiditis. It is more important in detecting the presence of intracranial complications. A normal blood picture in such a case means nothing but a pathologic picture is an infallible sign of complication although differentiation is not possible.

Balden<sup>10</sup> is in essential agreement with Heidemann. She states that infections of the ear have no specific blood picture and that differential diagnosis is possible only on the basis of marked changes in but few cases. Intercurrent infections, particularly grippe, obscure the findings; one must always take into account the age of the process, and the age and constitutional diathesis of the patient. Old lesions show a lymphocytosis, children have the same tendency, and certain patients, notably those with the so-called lymphatic diathesis, show the same thing. Gross disturbances usually register in the blood picture before clinical signs but in very acute severe cases signs and symptoms occur at once. Therefore, the blood picture is apt to be of more value in the borderline case where slight deviations sometimes offer operative indications. Temperature, blood picture and clinical course correspond well. One can differentiate between otogenous infections in degree only and not with any marked accuracy. The presence of a complication may be detected but not its type. The blood picture is corroborative but not a determining factor.

Glasscheib<sup>11</sup> is one of the few writers who feels that he can draw very definite conclusions as to the exact state of the pathologic process. In a study of 72 cases of suppurative mastoiditis he differentiated three groups:

1. Cases which had normal eosinophil percentage and a deviation index of 1-10 to 1-8 showed in the mastoid cells granulations but no free pus. (Deviation index is relation of immature cells to total neutrophils.)
2. Cases without eosinophils and with a deviation index as high as 1-3 showed in the cells free pus and granulations.
3. Cases with eosinophils increased from 3-6 per cent with a deviation index of 1-3 or 1-2 showed pus-containing cells in contact with the sinus. A thrombosis is very apt to show a

moderate grade of eosinophilia but one must rule out all other conditions which may cause eosinophilia.

Perisinus abscess shows a neutrophilia of from 85-90 per cent with a deviation index as high as 1-3. Sinus thrombosis shows an eosinophilia with a rise of the index to 1-2 or 1-1, and may be excluded when these two characteristics are absent; when it makes its appearance there is a specific decrease of neutrophils and leukocytes; therefore, the total leukocyte count is of no importance. Complications in the healing of the wound, such as phlegmon, erysipelas and abscess, do not show an index as high as 1-2. If the number of neutrophils rises to 85 or 90 per cent or if the index becomes reciprocal, with a low neutrophil figure, one may suspect septicopyemia. Meningitis of the convexity cannot be separated from that of other areas. Extradural and basal meningitis, as well as meningitis complicating cerebral abscess show no characteristic findings aside from neutrophilia; therefore, their presence must be determined by exclusion.

Piney<sup>12</sup> sums up the prognostic value of this laboratory aid as follows:

1. Slight neutrophilia, slight shift to left, and persistence of eosinophils may be physiological-pregnancy, mild infection.
2. Slight or definite leukocytosis, moderate nuclear shift (not more than 4 per cent young forms or 12 per cent band forms) and decrease of eosinophils or lymphocytes—no clear prognosis other than infection.
3. High leukocytosis, great shift to left, decrease or disappearance of eosinophils, and reduction of lymphocytes—always of grave prognostic import, especially when the percentage of young cells exceeds that of band forms.
4. Progressive fall of total leukocytes, extreme shift to left, total absence of eosinophils, and well marked lymphopenia, seen in moribund cases.
5. Progressive eosinopenia and increasing leukocytosis—infection to which reaction is occurring. Progressive eosinopenia and falling leukocytosis—infection to which no reaction is occurring.
6. Reappearance of eosinophils and decrease in leukocytes indicate improvement and is accompanied by shift to right.
7. Lymphocytopenia is always a bad sign.

8. Sudden fall in lymphocytes and progressive neutrophilia indicates extension of infection. Prognosis is bad when this occurs with diminution of total leukocyte count and shift to left.

9. Lymphocytosis following neutrophilia and eosinophilia with progression of neutrophils toward normal, both in number and "index" means recovery.

The consensus of opinion of men who have worked with the blood picture is that it is a valuable aid in determining the presence of intracranial complications but that it is of very little value in differential diagnosis.

#### BLOOD SEDIMENTATION.

The blood sedimentation test is a study of the settling power of red cells and is a general biologic reaction akin to fever. It has occupied the attention of investigators since the time of Galen—who called the buff colored stratum on the surface of a clot "*Crusta Phlogistica*." John Hunter, in 1791, and numerous other writers made observations concerning it. It dropped into disuse until Robin Fahraeus,<sup>13</sup> in 1917, working in the University of Kiel, rediscovered the phenomenon and reported that it might be used in the diagnosis of pregnancy, acute infection and malignant tumors. Since that time literature has abounded in reports of attempts made to apply it to practically every field of medicine and surgery. It is in no sense of the word a specific reaction or diagnostic test of any given condition but is an indicator of the presence and, more especially, of the progress of any condition which is attended by cell disintegration and destruction. In any such condition the sedimentation time of the red cells is much shortened.

An explanation of the mechanism of blood sedimentation is outside the scope of this paper. Guida<sup>14</sup> mentions among the theories which have been advanced for its explanation the following; that it is due to the constitution of blood plasma, the constitution of erythrocytes, the presence of megaloblasts, the presence of cholesterol and lecithin in the blood, and the more generally accepted theory that it is caused by modification of the colloidal state of the serum, that is, the altered relation between globulin and albumin.

A study of the literature reveals that the many technics described are all modifications of one or other of two original methods which depend on measuring the distance through which the head of the column of red cells falls in a given time, known as the distance method, originated by Fahræus and modified by Westergren so that it often goes by his name, or in measuring the time required for the head of the column of red cells to settle through a given distance, known as the time method, or Linzenmeier's method.

Westergren uses pipette tubes 300 mm. high and 2.5 mm. in diameter, with a mark 200 mm. from the bottom of the tube. The tube is filled up to the 200 mm. mark with a mixture of one part sodium citrate, 3.8 per cent, and four parts blood, and the height of the supernatant column of serum read at the end of one, two and twenty-four hours. The normal after one hour, for men, is about 3 mm. and for women from 5 to 10 millimeters.

Linzenmeier uses small tubes 5 mm. in diameter and 6.5 cm. high with five marks, one at the level of the height of a column of 1 cc. of water, and the others at 6, 12, 18 and 24 mm. below this. The tube is filled up to the cc. mark with the mixture described above and the time required for the head of the column to reach the 18 mm. line is determined. For normal men this is about 1200 to 1400 minutes, for women 800 to 1000 minutes. In menstruation the time is shortened, often to 600 minutes. Anything below 200 minutes is considered definitely pathologic.

Cutler<sup>15</sup> has described a graphic method which combines both time and distance factors and feels that this is important in that the rate of sedimentation varies in different stages of the process.

The test has been applied in many conditions, notably in gynecology and tuberculosis, in an effort to differentiate between acute and chronic infections, benign and malignant tumors, aseptic and septic abdominal lesions, nonhemorrhagic and hemorrhagic diseases. It has not proved a specific diagnostic test in the hands of most men but it has been found to be a valuable indicator of the presence of any process, toxic or septic, which is breaking down cellular tissue. Hence, sedi-

mentation time is apt to be decreased in pregnancy, sepsis, malignant tumors and hemorrhage. Workers in tuberculosis have found it of great value and some of them have reported that serial sedimentation tests give more accurate information than the pulse and temperature curve, weight curve, or physical signs and symptoms. The test has so far found but a limited use in otorhinolaryngology.

Weiss<sup>16</sup> found sedimentation normal in chronic otitis media, chronic mastoiditis with cholesteatoma and labyrinth symptoms and in furunculosis of the canal. Acute mastoiditis and acute otitis media gave decreased sedimentation time. He feels that it can play but a minor rôle in otologic diagnosis, especially in differentiation of intracranial complications, but that it may have some value as a guide to the efficacy of treatment and for prognosis.

Bertog<sup>17</sup> found decreased sedimentation time in acute infections of the middle ear; when used serially it is of value in doubtful cases in differentiating between surgical and nonsurgical cases.

Guida,<sup>14</sup> using the technic of Westergren, investigated a series of otologic cases; he finds that in acute infections of the middle ear, with or without mastoiditis, sedimentation is increased. In acute suppurative otitis media with mastoiditis, sedimentation reaches a high degree, which may vary from a normal of 20 mm. to a maximum of 109; when there is but little involvement of the mastoid sedimentation is less. In suppurative otitis media without mastoiditis, sedimentation is less, varying from 10 to 30 mm. the first hour. In bilateral, purulent otitis media with mastoiditis, sedimentation is increased, reaching 119 mm. in the first hour, while in the bilateral form without mastoiditis sedimentation is definitely less. In acute suppurations with increased sedimentation, operation is usually necessary; in other cases with bilateral disease and severe pain in the mastoid, when sedimentation is normal, recovery may occur in a few days. In cases of acute suppurative otitis media, convalescent, sedimentation is diminished. He concludes that in mild aural disease sedimentation is low and that when the disease is severe, requiring surgical intervention, sedimentation is increased. He finds very little differential value in the test.

## BLOOD DYSCRASIAS.

Numerous diseases have their first manifestation in the ear, nose or throat; the lesion may be an angina, adenitis, polyposis, gingivitis or membranous or ulcerative lesion in the oropharynx. This is especially true of diseases of the blood and blood forming organs, and it is in this class of cases that the differential blood count becomes of great importance in establishing an early diagnosis. The tonsillar swelling of leukemia, the gingivitis of hemophilia, the adenitis of Hodgkin's disease and the glossitis of pernicious anemia, acute leukemia, hemorrhagic purpura, arsenical poisoning, cachexia and severe sepsis are well known. These cases may come early to the otolaryngologist and he should realize the value of blood studies in their recognition. If he is wise he will enlist the aid of the internist in the exact diagnosis but he should himself always bear in mind the possibility of meeting them.

Agranulocytic angina and infectious mononucleosis are two diseases which are usually ushered in by malaise and angina and which, therefore, may be seen early by the otolaryngologist. They emphasize the importance of differential blood counts in obscure or undiagnosed lesions of the nose and throat, but it should be remembered that they are not the only diseases so announcing themselves.

The first case of agranulocytic angina, reported by Werner Schultz in 1922, has been followed in increasing numbers by others, most of them coming from Germany and Austria, with scattered reports from England, America and Italy. The disease is one of unknown etiology, some holding that it is a distinct clinical entity in which the unknown agent attacks the granulocytic elements of the bone marrow, others maintaining that it is a rare reaction to severe sepsis. It may appear at any age and in either sex, but is seen most often in middle aged females. It may or may not be preceded by other diseases. It runs the course of severe sepsis with increasing toxemia and terminates fatally in about 90 per cent of the cases, pneumonia being the most frequent terminal complication. The angina may be present from the beginning or appear later and may vary from the lesions of ordinary, acute, follicular tonsillitis to those of severe ulceration and necrosis, strongly sug-



gesting diphtheria. These lesions may be confined to the tonsil or may be seen on the gums, tongue, jaws and genitalia, and have been found at postmortem throughout the gastrointestinal tract. Jaundice is present in about half the cases; hemorrhagic diathesis is found in less than 20 per cent. Gross bleeding is rare, but petechiæ of the gums and mucous membranes have been seen. Cases without tonsillar involvement offer the best prognosis.

Postmortem has shown no special pathology in heart, lungs, kidney, liver or spleen, the findings in these organs being those of acute sepsis, e. g., cloudy swelling and fatty degeneration. The only distinctive pathology is that of the bone marrow, the tissue often being poor or lacking in granulocytic cells, while the red blood cell forming tissue is normal (so-called "red" marrow). Most of the cells present are lymphoid in nature. Reticuloendothelial cells are increased. Friedmann<sup>18</sup> observed fatty degeneration of the bone marrow in three cases. Section shows but few granular cells in the tissue around the ulcers, this being taken as evidence of the low resistance of the patient. Vincent's spirochete and fusiform bacillus, bacillus pyocyaneus, pneumococcus and streptococcus have been recovered from the mouth lesions. Blood cultures are usually negative, although streptococcus, pneumococcus, staphylococcus, streptococcus viridans and bacillus pyocyaneus have been found. The opinion has been expressed that such findings should not be taken as proof of the specific nature of the disease, inasmuch as one may get positive blood cultures in the terminal stages of various diseases, notably lymphatic leukemia.

Diagnosis is made from the differential blood count. This shows an absolute diminution in the number of both granular and lymphocytic cells, most marked in the former, so that a relative lymphocytosis exists. The total white count may vary from 200 to 4,600, and the granular cells may be absent or so scarce that monocytes, mast cells, and eosinophils cannot be counted. Red cells are normal, or slightly diminished, blood platelets normal or above normal, and hemoglobin normal.

No adequate therapy has been found. Transfusion, X-ray, diphtheria antitoxin and arsphenamin have been used without marked benefit.

A complete bibliography may be found in the extensive review of Rose and Houser.<sup>19</sup>

Several diseases are characterized by lymphocytosis, among them being acute lymphatic leukemia, various infections, such as whooping cough, and a third one, infectious mononucleosis, which is very apt to come to the laryngologist because angina is an early and constant symptom. Its exact identity has not been determined. Filatow<sup>20</sup> regarded it as an idiopathic cervical adenitis; it has been considered by many writers as identical with the so-called glandular fever first described by Pfeiffer.<sup>21</sup> It was established as a clinical entity in this country by the papers of Sprunt and Evans,<sup>22</sup> and Longcope.<sup>23</sup> Downey and McKinlay<sup>24</sup> have called it acute lymphadenosis, and also regard it as a clinical entity. It is essentially a disease of young adults, most often males, is always accompanied by angina, and presents in the blood a cell which serves to differentiate it from the other lymphocytoses.

There is no known predisposing cause or etiology. It is announced by the symptoms of any acute infection, e. g., headache, fever, chill, sore throat, and malaise, usually mild. The angina is constant and early. Physical examination shows various types of throat lesion. There may be only an intense injection of the fauces, or there may be scattered patches of exudate or actual ulcerative lesions of the tonsils. There is present a general adenopathy, most marked in the upper anterior cervical area, but involving also the axillary, epitrochlear and inguinal glands. Splenomegaly is seen but no liver enlargement. Hemorrhages are rare, although petechiæ occur in the buccal mucosa. Purpuric rash has been reported. The affection is always a mild one and terminates in recovery, usually in two or three weeks.

The differential diagnosis must consider especially acute lymphatic leukemia and infectious lymphocytosis, and is made on the differential blood smear. There is present a moderate leucocytosis, the count ranging from 11,000 to 26,000, and a marked lymphocytosis, the percentage of these cells varying from 57 to 92 per cent. The red cells and hemoglobin are normal or at most show only a moderate secondary anemia.

Whereas most types of lymphocytosis show an immature or lymphoblastic type of cell, infectious mononucleosis shows

a mature, fully functioning, but abnormal lymphocyte, which, according to Downey, is so characteristic that its presence makes the diagnosis.

The blood differential must rule out especially acute lymphatic leukemia. Early clinical conditions may be very similar but the good outlook in one and the fatal prognosis in the other make an early differentiation imperative. Acute lymphatic leukemia is characterized by a higher white count, often 50,000, by the time angina occurs, and the cell present is an immature or lymphoblastic cell instead of the mature but abnormal cell of infectious mononucleosis. There is present also a marked anemia, not to mention clinical signs which render recognition possible.

Downey and McKinlay<sup>24</sup> have done the most exhaustive work in the cytology of the disease, and Cady (*loc. cit.*) gives an excellent bibliography.

#### BACTERIOLOGY OF THE BLOOD.

The bacteriology of the blood has long been studied in general medicine, and septicemia complicating systemic disease, such as typhoid fever, severe pneumonia and terminal erysipellous infection has long been recognized. The importance of blood cultures in the study of infections of otitic origin was called to the attention of the profession about 1906 by a brief report by Libman<sup>25</sup> to the American Otological Society. This aroused great interest and was followed by much work, until the procedure was placed on a definite basis and given a recognized position among the diagnostic procedures in otology. A study of the papers which appeared at that time and in the immediately succeeding years gives us most of the information which we now possess concerning the value of this procedure. Particularly important are those of Libman and Celler, Gruening, Sondern, Page, Leutert, Oppenheimer and Hayes, all of which are quoted by Libman<sup>26</sup> in his exhaustive article in the Transactions Ninth. Internat. Otological Congress, Boston, 1912.

As a result of the work which was done it became apparent that a bacteremia may be demonstrated in a certain percentage of aural complications and that this bacteremia is most often

seen in sinus thrombosis and meningitis. The question was raised whether or not a bacteremia was present only in complicated otitic infections or whether this finding might also be obtained in uncomplicated cases of acute suppurative otitis media and mastoiditis. Most of the workers obtained positive blood cultures only in cases of mastoiditis complicated by sinus thrombosis or meningitis. Duel and Wright,<sup>27</sup> however, obtained positive blood cultures in cases of uncomplicated mastoiditis which presented no sign of sinus thrombosis but subsequent workers were unable to duplicate their results.

Cases are frequently reported in which clinical sepsis and positive blood cultures found in aural infections clear up after jugular surgery without the demonstration of a thrombus, and the explanation has been advanced that the bacteremia may be caused by thrombophlebitis of small tributary radicals such as the minute veins in the mastoid septa or by phlebitis of a major vessel with erosion of the intima but without actual thrombosis. Kerrison<sup>28</sup> describes an infectious sinus phlebitis without demonstrable clot, with erosion of the intima, and with periodic invasion of the blood stream. Crane<sup>29</sup> states that "it is obvious that we have a goodly number of cases of otitic sepsis due to invasion of the blood stream through channels other than the sigmoid sinus and that we have, often enough, a thrombophlebitis of the smaller veins in the temporal bone resulting in a bacteremia without gross evidence in the large sinus." Eagleton<sup>30</sup> states that "thrombophlebitis of small venous radicals within the cranium is not associated with positive blood cultures; and although cases of positive streptococci blood cultures from thrombophlebitis of small venous radicals in other parts of the body are reported, in infection from the ear a positive blood culture should be regarded as diagnostic of phlebitis of a large venous sinus." The difficulty in such cases is to rule out a small mural or bulbar thrombus, undetected at operation or autopsy, and the remarks of Libman,<sup>26</sup> in 1912, are still pertinent: "The general conclusion one can draw from the literature on clinical and experimental investigations above presented is that while there seems to be a certain amount of evidence that general infections may occur without involvement of the sinus such an occurrence is so infrequent that it plays no rôle in actual practice and that one has no way of

knowing that a given case is the exceptional one in which a thrombosis is not present."

Blood culture has a secure place in otology, even though writers regard it somewhat differently. Lillie<sup>31</sup> says that "blood culture is far more important than a leukocyte or differential count." Kopetzky,<sup>2</sup> on the other hand, feels that too much reliance has been placed on blood cultures; that it is a mistake to wait for them, and that more attention should be paid to other phases of the blood picture. Eagleton recommends daily blood cultures, saying that while one or a series of negative reports means nothing, one positive report means everything, and Dench<sup>32</sup> states that a "positive blood culture in a case of suppurative otitis with symptoms of sinus thrombosis renders the diagnosis absolutely certain."

Blood cultures may be positive in diseases such as erysipelas, angina, endocarditis, pneumonia or typhoid fever. The first three are most apt to complicate an ear lesion. After efficient blood vessel surgery continuation of a positive blood culture means either that the organisms are multiplying in the blood or that an endocarditis is present.

The consensus of opinion is that a positive blood culture, all other causes being excluded, means thrombophlebitis of a major vessel, and is to be taken as an indication for isolating the ear from the general venous circulation.

Libman<sup>26</sup> gives very concisely the causes for a negative blood culture in the presence of thrombosis. The organisms may be too few in the specimen taken, improper or incomplete cultural methods may be used (both aerobic and anaerobic organisms should be sought), the cultures may be made too soon, or they may be made after the discharge of organisms from the local focus has stopped, the bacteremia may be intermittent and the culture made at the improper time, the blood may be so bactericidal that the organisms do not live for any length of time, the ends of the clot may be sterile, the thrombus may be infected but may completely block the vessel, or the thrombus may be sterile.

A consideration of these facts explains the statement that one or a series of negative blood cultures does not rule out sinus thrombosis and that in the presence of aural sepsis it is

not wise to wait for a positive culture before resorting to surgery.

Septicemia complicating rhinologic conditions is not common, although Libman<sup>26</sup> states that positive blood cultures may be obtained in cases of severe accessory sinus disease and nasal infection. The bacteremia which complicates infections of the upper lip and vestibule of the nose are well known, as is also the cavernous sinus thrombosis which not frequently ensues in such a situation. One finds an occasional case report of bacteremia from organisms commonly found in the nasopharynx, such as *micrococcus pharyngitidis sicca*.<sup>33</sup> Such findings, however, are infrequent when compared with those obtained in aural sepsis.

Early and transient bacteremia offers the only explanation for the sequelae of acute angina. Tanaka and Crow<sup>34</sup> state that "in ordinary chronic tonsillitis the lining epithelium of the crypt is replaced with scar tissue. Occasionally definite ulcers are found. In other cases large numbers of blood vessels have been thrombosed and they afford a path for the entrance of bacteria into the blood stream." Ballenger<sup>35</sup> goes into the question of bacteremia associated with acute hemolytic streptococcus throat infections and states that "the fact that complications of acute angina are due to blood stream infection is becoming increasingly evident" and mentions among such complications peritonitis, appendicitis, deep wound infections and acute thyroiditis. He feels that a bacteremia secondary to an acute throat infection is probably a common occurrence and possibly is the usual accompaniment of this type of infection. He also feels that with an acute otitis media or an acute mastoiditis complicating an acute streptococcus angina a positive blood culture of hemolytic streptococcus is not of as great diagnostic import, so far as thrombosis of the lateral sinus is concerned, as are cultures of other pus producing organisms. Claus<sup>36</sup> reports a series of twenty-eight cases of postanginal pyemia with hemolytic streptococcus bacteremia in several; various organisms have been isolated from the blood in aggranulocytic angina.

#### BLOOD CHEMISTRY.

Blood chemistry, while of but limited value in diseases of the ear, nose and throat, is frequently necessary, particularly

the study of blood sugar in surgical cases. This should be made in all diabetics, especially those coming up for operation, and the patient prepared by careful feeding and the administration of insulin. This, however, should be done under the supervision of an internist. Hemorrhagic mastoiditis is apt to progress rapidly and painlessly in a diabetic (Kopetzky<sup>2</sup>), and early operation may be necessary.

Estimation of blood calcium has been used by Kopetzky and Almour<sup>37</sup> in the study of undifferentiated deafness. These authors studied a group of cases of middle ear deafness in which the diagnosis of otosclerosis was not certain. They found that the greatest number of patients with diminished blood calcium coincided with those having a true otosclerosis, and that altered calcium content was associated oftener with heredity and raised lower limit than with prolonged bone conduction, history of infection or pregnancy. In eleven out of twelve otosclerotics blood calcium was altered. They concluded that altered calcium metabolism is corroborative evidence of otosclerosis in a patient with deafness and heredity, and that when the deafness began with infection or pregnancy and the patient has an elevated lower limit and prolonged bone conduction, either increase or diminution of calcium content, and particularly the latter, suggests otosclerosis.

Vermes and Brügel<sup>38</sup>, in an extensive study in Neumann's Clinic, made the following observations: Blood calcium in operated and nonoperated cases of acute suppurative otitis media and acute exacerbations of chronic suppurative otitis media usually shows some increase, which fluctuates, and is dropping at the time when surgical indications are present. Increased calcium content is found after bone trauma, such as mastoidectomy. Nonsuppurative lesions of the middle ear show normal findings. Cases coming to operation late are apt to show normal calcium content because the destructive stage has passed and reparative processes are appearing. These findings are explained by the fact that bone inflammation causes increased absorption of calcium. No operative indications can be drawn from the study of blood calcium.

Novak,<sup>39</sup> in a study of hyperesthetic rhinitis, found some of the patients to have a hypocalcemia but does not believe this to be a constant or important finding.

Friesner and Rosen<sup>40</sup> report a series of cases in which the calcium content of the pus from lesions in bone and in soft tissue was analyzed, using the method of Kuttner, and Kuttner's colorimeter. In general, they found that the pus showed more calcium when there was a destructive bone process, abscess of soft parts showing an average of from 7 to 9 mgs. of calcium per 100 cc. of pus, and disease of the bone showing an average of from 18 to 33 mgs. per 100 cc. They found the method a helpful guide but could derive no operative indications from it.

#### BLEEDING AND COAGULATION TIME.

Bleeding and coagulation time are of interest on account of their relation to surgical procedures; their value has been questioned and reports indicate a wide divergence of practice in their application.

Bleeding time is the time during which bleeding occurs from a stab incision made with a small lancet or needle, either in the lobe of the ear or the finger. Osler<sup>41</sup> states that it makes no difference whether the cut is short or long or whether, within certain limits, it is superficial or deep. Duke's method is widely used; the incision is made and a drop taken up on blotting paper at regular intervals—usually 30 seconds—until bleeding stops. If this occurs within five or ten minutes one may assume slight prolongation of bleeding time; when the twentieth drop is one-half the size of the first there is moderate prolongation. If the twentieth drop is as large as the first there is great prolongation. In a questionable case the test should always be made at more than one place in the skin, and at different times, on account of the variation which the case may present.

Bleeding time does not necessarily parallel coagulation time; it depends on numerous factors, such as the presence of tissue juice (thromboplastin), elasticity of the skin, sclerosis of the vessels and the mechanical and chemical action of blood platelets. Hemorrhage is controlled by the formation of platelet thrombi in the injured capillaries.<sup>42</sup> When the platelets are reduced, as in purpura hemorrhagica, bleeding time is prolonged. This is explained by the fact that the clot keeps the tissue juice from reaching the open vessel and by a peculiarity



of the platelets which prevents their normal agglutination and disintegration. Normal bleeding time is from one to three minutes but may be as long as eight. It is prolonged in severe anemias, when the fibrinogen content is low (chloroform or phosphorus poisoning) and in destructive bone disease. In thrombopenic purpura (*purpura hemorrhagica*) it is greatly increased. In all other purpuras it is normal. Some writers have found it normal in hemophilia, others prolonged, but rarely as long as in *purpura hemorrhagica*, being usually less than eight minutes. Duke found it normal in jaundice.

Coagulation time is the time required for the blood to clot outside the vessel; it is influenced by the temperature of the container, size of the drop, smoothness and cleanness of the instrument, and the method which has been used in withdrawing the blood. Blood taken from a puncture wound will contain some tissue juice and will have a shorter coagulation time than blood drawn by venipuncture. Howell<sup>43</sup> gives coagulation time as twenty minutes (thirty to forty by venipuncture), and Lee and White<sup>44</sup> as ten minutes; their methods with venipuncture are widely used.

Coagulation time is important only when it shows marked prolongation as in hemophilia; Osler states that such variation is the only constant pathologic sign of this disease. Slow coagulation is due to delay in the formation of thrombin, not to the action of thrombin or fibrinogen. In any case in which there is a question of hemophilia venipuncture should be used; by this method clotting time may vary from four to forty minutes; when obtained from an incised wound it may vary from five to nine minutes. Coagulation time is prolonged in melena neonatorum, obstructive jaundice, some of the anemias, leukemia, and some of the severe infections such as pneumonia. It would appear that bleeding time is of greatest value in thrombopenic purpura (*purpura hemorrhagica*), and possibly in hemophilia, although it may be normal in the latter condition, and that coagulation time is of particular value in true hemophilia.

The relation of coagulation time to postoperative hemorrhage has prompted several recent reports. Kleinert<sup>45</sup> found routine coagulation time of little value and thought there was no particular relation between prolonged coagulation time and postoperative hemorrhage. Other things, such as the age of

the patient, blood pressure, sclerosis of the vessels, and personal and family history are of more importance in warning the surgeon of hemorrhage. McKinney<sup>46</sup> thinks that the test is of no special value but should be done as a matter of protection in case any question arises, and to satisfy the requirement of the patient. Many of them know that such a test is frequently done and do not feel that all precautions are being taken unless it is made. The normal time varies from one to eight minutes; the test would be more valuable if we had a more reliable technic. Hunt<sup>47</sup> found from a questionnaire that the capillary tube method is the one most commonly used in the determination of coagulation time, that the test has but little bearing on postoperative hemorrhage, that it is not necessary if a proper history has been taken and a careful physical examination made, but that in all doubtful cases both clotting and bleeding time should be done. His report would indicate a wide variation in this practice, many surgeons always using it, others never using it, and others using it in selected cases. It appears from a study of all reports that coagulation time under fifteen minutes has no particular relation to postoperative hemorrhage and that in cases where the tests show the most variation, bleeding time in purpura hemorrhagica, and coagulation time in hemophilia, the diagnosis can be equally well established by physical examination and history.

WASSERMANN.

The following statements relative to the practical value of the Wassermann reaction may be made:<sup>48</sup>

1. It is of special value in extragenital sores and atypical lesions of all stages.
2. A negative reaction is of less value than a positive.
3. In early latent syphilis after the chancre has healed and before secondary lesions have appeared the Wassermann is the only diagnostic test.
4. It is often the only diagnostic aid in late and tertiary syphilis.
5. It may be negative in the blood serum and positive in the spinal fluid in cases of cerebrospinal syphilis.
6. A persistently positive reaction means the presence of living spirochetes in the body.

Syphilis occurs not infrequently in otolaryngology and the Wassermann reaction should be used freely in attempting to clear up the diagnosis of obscure lesions. Fournier<sup>49</sup> found 850 extragenital chancres in 10,000 cases of primary syphilis, and of these 75 per cent were in the region of the head, mostly about the mouth and throat. Fifty per cent of all extragenital chancres were on the lips.

St. Clair Thomson<sup>50</sup> has this to say about the occurrence of syphilis in the nose and throat: "Primary syphilis of the nose has been diagnosed but is rare. Secondary syphilis of the nose is almost unknown, being most commonly seen as the "snuffles" in infants suffering with hereditary lues. It may give a very persistent nasal catarrh in adults, which resists all treatment and does not respond to cocaine. Mucous patches in the nose are rare."

Tertiary syphilis of the nose is found in the form of gumma, which is the foundation of all manifestations of that stage which appear in the nasal cavity, such as typical gumma, with or without ulceration, perichondritis, with or without necrosis, and certain types of atrophic rhinitis. The Wassermann should be used in cases of atrophic rhinitis, perforation of the vomer, ulcerative processes, sequestra, or perichondritis. It is a good preoperative measure in certain patients who are to have nasal surgery, particularly submucous resection, especially when there is any suggestion of syphilis. Such surgery should not be done if there is any evidence of an active process in the nose. Occasionally a Wassermann test will reveal the cause of delayed healing after submucous resection when the nose fills up with a gummy, adhesive process. Such cases may clear promptly under specific treatment.

Secondary syphilis of the larynx manifests itself usually in the form of erythema or mucous patches. Tertiary syphilis of the larynx takes the form of gummatous infiltration with or without ulceration. Due to the fact that syphilis of the larynx may occur with or without tuberculosis and carcinoma, the Wassermann reaction in a suspected lesion of the larynx does not have an absolutely conclusive significance. A positive Wassermann in the presence of such a lesion means, as before stated, that the patient is syphilitic. It does not necessarily mean that the lesion under consideration is luetic. St. Clair

Thomson, in summing up the situation, says: "The Wassermann reaction is helpful but not conclusive."

In otology the indications for the Wassermann test are perhaps more limited than in rhinology and laryngology. Rarely ulcerative lesions of the canal or auricle are luetic.<sup>51</sup> The rôle of syphilis in diseases of the internal ear is well known; suffice it to say that any case of sudden profound deafness, with marked diminution of bone conduction and depression of the upper limits of hearing, or any case of sudden, severe tinnitus and vertigo should have a Wassermann. It should be done in cases of congenital deafness.

#### CEREBROSPINAL FLUID.

The normal pressure of the cerebrospinal fluid is always the same as the general intracranial pressure as measured in the torcular herophili and varies from 60 to 150 mm. of cerebrospinal fluid, being greater in the ventricles than in the subarachnoid space. Increased pressure is seen in cases of cerebral tumor, hemorrhage, hydrocephalus, meningitis and encephalitis; it may reach 500 or 1,000 mm. of fluid. Low readings are found when the fluid in the spinal column is not in full communication with that within the cranium, as in spinal tumor, Pott's disease, spinal and cerebrospinal meningitis with adhesions, or in spinal compression.

Changes in appearance may be due to the presence of color or turbidity, neither of which is ever present in a normal fluid. Coloring varies from a faint, pale yellow to a deep chrome. It is seen in meningitis; a yellow color is constantly present in subarachnoid hemorrhage.

Turbidity may vary from a slight trace to that of thick pus. When only a trace is present one must rule out chemical coagulants, such as carbolic acid or alcohol, which may have been present in the needle, or traces of blood. Turbidity is caused by the presence of cells, organisms or fibrin web. Neutrophils are more likely to cause turbidity than lymphocytes, but the clouding depends on the death and degeneration of cells rather than on their nature. A count of over 300 neutrophils usually gives an obvious turbidity, while one may have over 500 lymphocytes with a clear fluid. Cloudiness is no indication of the severity of the infection.

The fibrin coagulum, which is the most delicate test for fibrinogen, is often missed because the fluid is shaken up in transport to the laboratory; when a fluid is being examined a small portion should be saved with the object of watching for the formation of the web. This may vary from a fine, delicate skein to solidification of the whole tube and is particularly apt to be found in tuberculosis and syphilis, especially with highly albuminous fluids. Any increase in protein above 100 mgms. per 100 cc. may be accompanied by fibrin coagulum; it may not form, however, owing to the absence of the fibrin ferment, even in the presence of considerable fibrinogen. This may be supplied by the addition of a drop of fresh blood or serum, when coagulation will occur.

Neutrophils are not found in normal fluid but even a trace of blood will cause them to appear. They are seen in pathologic conditions, especially acute infections, and are apt to dominate the picture in acute coccal meningitis and cerebral abscess; they may constitute 50 per cent of the cells in tuberculous meningitis and poliomyelitis. Lymphocytes are found particularly in syphilitic conditions and outnumber the other cells except in acute epidemic, coccal meningitis and brain abscess. Large mononuclear cells (endothelial cells) are always present in pathologic fluid but never constitute more than 10 per cent of all cells. Eosinophils are present in small numbers in acute meningitis. Plasma cells are characteristic of chronic diseases of the cerebrospinal system. Compound granular corpuscles are seen in cerebrospinal lues, and tuberculous meningitis; macrophage cells occur in subarachnoid hemorrhage and tumor but have no special pathologic significance.

The normal fluid contains very few cells. Nonne gives 5 per cubic mm. as normal. Other writers say that three is the average and that anything above four is pathologic. Greenfield and Carmichael<sup>52</sup> classify pleocytosis as follows: 5 to 10 per cubic mm.—slight, 10 to 50—moderate, 50 to 250—severe, and above 250, extreme.

These authors divide the cytologic changes into three types, the first, in which are found lymphocytes and endothelial cells of the so-called mononuclear type, the second, in which is found the mononuclear cell with some pathologic cell, such as the

neutrophil, and the third, which is essentially neutrophilic; they point out that with slight reaction the lymphocyte is the cell most apt to be found, with severe reaction (50 to 250 cells), both the mononuclear and the neutrophil, and in the very severe type the neutrophil.

The estimation of protein has been of value in measuring inflammatory changes in the meninges. The two proteins most commonly found are globulin and albumin, the latter present in the greater amount; there is no known condition in which globulin exceeds albumin. A mild or moderate increase in protein is the commonest chemical change in the cerebrospinal fluid; a great increase is rare. The normal content is from 25 to 100 mgm. per 100 cc.; increase up to 500 mgms. is seldom seen; content above 500 mgms., rare. Increased protein is apt to occur as an isolated phenomenon or with altered color and increase of cells, and is characteristic of brain tumor, cord tumor, cord compression and localized spinal meningitis. Increase of protein with increase of cells is the commonest pathologic finding and is seen in all forms of meningitis, acute, subacute and chronic, and in various other lesions, but the protein increase is not always parallel with the cell increase. In general, in meningitis the protein does not rise above 200 mgms. per 100 cc. of fluid; rarely it goes to 750, 2,000, or even 3,000 mgms. Such fluids, with scant or absent cells suggest a meningitis limited to the upper part of the cord and base of the brain.

Glucose is normally present in amounts varying from 45 to 85 mgms. per 100 cc. The blood contains more sugar than does the cerebrospinal fluid and its sugar content varies more rapidly with physiologic and pathologic conditions than does that of the spinal fluid. Increased glucose content in spinal fluid is seen as an accompaniment of increased blood sugar in diabetes mellitus and acute nephritis, and may reach 100 mgms. per 100 cc. in many general diseases. Diminution is a cardinal sign of acute meningitis, whether tuberculous or coccal, but is usually slight in the early stages; in late stages sugar may be absent. This is due to the presence of ferments which result from cellular disintegration, or of organisms which use glucose as food. The variation in glucose is most important in acute meningitis.

Chlorides are normally present in from 720 to 750 mgms. per 100 cc. of spinal fluid. This figure is so constant that any variation is regarded as pathologic. Any increase above 750 mgms. is indicative of renal deficiency; figures lower than 720 mgms. are always important and usually pathologic. Certain conditions, such as brain tumor and cerebral abscess, may give a cytologic picture much like acute meningitis, which may be ruled out by normal chloride content. A progressive lowering of the chlorides means invasion of the subarachnoid space; in acute meningitis the figure may drop to 650 mgms. per 100 cc. In tuberculous meningitis particularly, the diagnostic value of the chloride content is second only to that of the presence of tubercle bacilli. The chloride in cerebrospinal fluid never falls below the blood chloride; progressive lowering or a single low reading is a bad sign.

Increased meningeal permeability appears in pathologic conditions and has been made use of as a diagnostic aid. Sodium nitrate particularly has been so used. One gram by mouth for each 30 kilos of body weight should normally show from 1 to  $1\frac{1}{2}$  mgms. per 100 cc. of spinal fluid in three hours. In meningitis this may be increased to 5.5 mgms. and in tuberculous meningitis to 8.5 mgms.

Kafka, in 1912, found that uranin (sodium fluorescein) by mouth in 6 to 8 gram doses appeared in the spinal fluid in 30 per cent of nonsyphilitic and 100 per cent of paralytic cases. In the majority maximum elimination was at the end of three hours, but traces appeared anywhere from one to seventeen hours.

Most writers describe a serous meningitis, a suppurative meningitis, and a condition known variously as meningism or meningismus. Suppurative meningitis is divided into circumscribed and diffuse, and the same classification is occasionally found in the descriptions of serous meningitis.

By serous meningitis is meant an inflammatory condition of the arachnoid or pia, usually secondary to some lesion, such as extradural abscess, in which these membranes, though congested and edematous, have escaped actual invasion, there being present secondary edema but no pus or bacteria (Kerrison<sup>28</sup>). Politzer<sup>51</sup> describes a serous infiltration of the pia mater caused

by inflamed meninges and accompanied by an increase in the amount of cerebrospinal fluid. Greenfield and Carmichael<sup>52</sup> state that the term includes a great variety of mild infections of the meninges due to the presence of avirulent organisms in the subarachnoid, or a more virulent organism in the layers of the dura or the subdural space. Kerrison states that in purely serous meningitis organisms and pus cells are not usually found, but Yerger,<sup>53</sup> in a recent study, found organisms in 30 per cent of the cases with a cell count of 250 or less. In this type of meningeal involvement the fluid is usually clear, but on occasion may be cloudy; there is usually no color and the pressure is increased. The count is usually low (below 250 per cubic mm.) and the cells of the lymphocytic type, although the neutrophil may predominate. Globulin is increased; sugar is diminished or absent.

By circumscribed, suppurative meningitis is meant a process limited by marginal adhesions which prevent a diffuse meningitis but do not keep organisms from reaching the spinal canal. The fluid may yield both cells and organisms and "the presence of pus and bacteria in the fluid may constitute no evidence of the existence of what may properly be called a diffuse, purulent meningitis" (Kerrison). Politzer describes a leptomeningitis circumscripta, which is a walled-off collection of pus between dura and pia, and Yerger mentions a circumscribed, suppurative meningitis which includes the cases of so-called protective or sympathetic meningitis. The fluid is turbid or frankly purulent and may present a faint greenish yellow or yellow color; the pressure is increased. The cells vary from 250 to 5,000 (Yerger<sup>53</sup>). He makes the cell count one of the criteria for differentiation of circumscribed from diffuse meningitis, counts under 5,000 usually indicating a circumscribed process and those above 5,000 a diffuse process. The cell predominating is the neutrophil. The total protein may vary from 100 to 200 mgms. per 100 cc. Globulin is increased. Sugar is reduced or absent and the chlorides may be reduced as low as 600 or 650 mgms. per 100 cc.

Diffuse, suppurative meningitis is a general invasion of the subarachnoid space by the infectious process. Some authors do not divide such a process into circumscribed and diffuse, but such a classification is usually found. The basis of classifica-



tion depends on the cell count and the severity of the bacterial invasion, low counts pointing toward a circumscribed process and high counts indicating a diffuse process. Yerger places cases with a cell count of 5,000 or under in the circumscribed class and those with 5,000 or more in the diffuse, and states that the presence of bacteria in cerebrospinal fluid is the best clinical evidence of a diffuse meningitis. Kerrison disagrees and says that the presence of pus and organisms does not necessarily mean a diffuse, purulent process. Greenfield and Carmichael state that a large number of organisms in smear and culture indicates an impending diffuse meningitis and that diminution of sodium chloride to 680 mgms. in a turbid fluid has the same significance. Cell counts up to 1,000, protein increase up to 100 mgms. and absence of glucose do not necessarily mean diffuse meningitis as long as the chlorides remain normal. A fluid containing only a few avirulent organisms does not necessarily mean a diffuse process. Most reported cures of meningitis are those classified as circumscribed, with or without organisms, and with low cell counts, often predominately lymphocytic.

The condition known as meningismus usually presents a fluid normal except for increased pressure.

The fluid in brain abscess may be perfectly normal or it may present the picture of a circumscribed meningitis with a sterile fluid which may be either serous or purulent. The count may be very low, under 10, and predominately mononuclear; Yerger found it to be under 500 in 95 per cent of his cases. Eagleton points out that the count is apt to be high in the early stages, dropping later as the process becomes walled off, when it may be as low as 250. The pressure is increased, globulin may be present and sugar diminished or absent. The spinal fluid findings are influenced by the proximity of the abscess to the subarachnoid space. Normal fluids may be found, as before stated, if the abscess is deeply seated and well walled off. If it is near the surface the fluid is apt to show the changes characteristic of the complication accompanying the abscess. In localized processes, such as extradural abscess, the picture is apt to be that of serous meningitis or circumscribed suppurative meningitis; if true diffuse meningitis is present, then the fluid findings will be those of the latter process.

It is important that the spinal fluid be studied in conjunction with other signs and symptoms. A brain abscess may have a perfectly normal fluid or it may present the picture of a circumscribed meningitis. A serous meningitis may or may not have organisms as may also diffuse, suppurative meningitis. The cell count, while giving us some information, does not definitely classify the spinal fluid. In general it may be said that serous meningitis is apt to have a clear fluid under pressure with a low count, predominately lymphocytic, and that suppurative meningitis is more apt to have a turbid fluid with a high count, essentially neutrophilic, and with a greater number of organisms present.

Lumbar puncture should be done daily in cases of suspected intracranial involvement and in the postoperative care of brain abscess in which it gives us our most exact knowledge of the changes going on in and about the lesion. It is well to do lumbar puncture before operation in chronic, suppurative otitis media; it may reveal the presence of an unsuspected complication.

In 1925 Dowman<sup>54</sup> described compression of the jugular veins in the diagnosis of thrombosis of the lateral sinus; Tobey and Ayer<sup>55</sup> reported in greater detail their experience. The procedure is based on the work of Queckenstedt and depends on variation of pressure of cerebrospinal fluid. Absence of rise of fluid pressure on jugular compression may indicate spinal subarachnoid block, as seen in cord tumor and tumor of the cerebellar fossa, or venous blocking, as seen in sinus thrombosis.

The test is made by doing lumbar puncture, using either a water or mercury manometer, and then gradually compressing first one and then the other internal jugular, noting the variations recorded in the fluid column. In thrombosis the pressure will be increased two or three times normal when the patient's jugular is compressed, while pressure over the thrombosed vein causes no rise or a slow rise of from 10 to 20 mm. This is usually indicative of complete obstruction. Partial obstruction gives less striking results.

The procedure occasionally shows no rise of pressure when obstruction is absent. This is usually attributable to the fact that the pressure is abnormally low or that the jugulars are

inaccessible. Low pressure may be corrected by the injection of 10 to 20 cc. of salt solution when the test may show normal results. Abnormal increase of pressure in the absence of obstruction is usually due to carelessness in making the test. The authors found that a difference of 50 mm. between the two sides was unusual and one of 100 mm. was exceptional; with a normal initial pressure (150 mm.) the rise will seldom be less than 50 m. and more likely will be over 100.

Anatomic variations in the veins explain slight rise of pressure sometimes obtained after compression of the thrombosed side. The test must be carried out under exact technic. Death from medullary pressure is possible; acute meningitis precipitated by the procedure was not noted.

Gaillard and Mayoux<sup>56</sup> consider the test an excellent means of diagnosis in block of the subarachnoid space. Not only must the subarachnoid space be free but the nervous system must be normal in order to demonstrate normal resistance to jugular compression. Elevation of pressure may transmit itself from the jugular to the cerebrospinal fluid by anastomoses between the lateral sinus and the internal jugular which are insufficient in most cases to render the test inaccurate. These authors do not feel that compression of the cervical sympathetic plays any part in the result. One case is reported in which the test was positive, although the sinus was normal, and one in which the test was negative in the presence of a thrombosed sinus. In the first instance increased pressure was due to compression of the sinus by a perisinus abscess. Such pathology, however, does not always give a positive reaction. They conclude that the test is of definite value in diagnosis of thrombosis of the lateral sinus and recommend its systematic use, bearing in mind the possible causes of error.

Aubry<sup>57</sup> reported a case of obliterative thrombophlebitis in which the test was negative; this was explained by the slow evolution of the occluding process and by coincident serous meningitis which had persisted for a long time after disappearance of the focus of infection.

Mayoux<sup>58</sup> concludes that Queckenstedt's test is of great interest in the diagnosis of thrombosis of the lateral sinus and should be included in daily practice.

## BACTERIOLOGY.

A great many studies have been made of the bacteriology of acute and chronic infections of the middle ear; the streptococcus hemolyticus, the pneumococcus, the streptococcus mucosus (pneumococcus type III), and the staphylococcus, have been named as the most important, with a long list of other organisms which includes the diphtheroid bacillus, bacillus proteus, bacillus pyocyaneus and bacillus coli. The exact relative importance of the first group has varied somewhat with different writers but in recent literature the most importance, particularly as regards the severity of the infection and tendency to produce intracranial complications, has been attributed to the streptococcus hemolyticus and streptococcus mucosus, with the other organisms playing a minor rôle.

Bacteriologic studies of acute ear infections are best made at the time of paracentesis or spontaneous rupture, under conditions which as far as possible avoid contamination from the skin of the canal; the organism responsible for the pathology can be recovered in most cases. Contamination soon occurs and the primary organism may be overgrown by invaders so that cultures taken from an acute otitis media of several days' duration are much less reliable in telling us the organism primarily responsible. This is true also of cultures taken in chronic, suppurative otitis media in which contamination has long existed so that the original infection has been overgrown and perhaps lost. Cultures taken under such conditions have very little value.

One of the best recent studies of the bacteriology of acute otitis media is that of Valentine,<sup>59</sup> who followed a series of acute middle ear infections over a considerable period of time and attempted to correlate the changing bacteriologic picture with the varying clinical appearance. She found, as have most other writers, that the original infection is monobacillary; after a few days it becomes mixed, due to contamination. Beta hemolytic streptococcus was the probable cause of infection in over 50 per cent of her cases; there was marked correlation between the bacteriology of the throat and ear when the beta hemolytic streptococcus, the green streptococcus, or the pneumococcus was found in the aural secretion. The green

streptococcus is found in normal throats. Beta hemolytic streptococcus is easily overgrown by staphylococcus and diphtheroid bacilli, and normal throat inhabitants, such as the green streptococcus and gram negative coccus, come to the foreground. This has been mentioned by other writers who have referred to the disappearance of the secondary invaders under treatment and the later reappearance of the primary organism with exacerbation of symptoms. Ninety per cent of the cases showing beta hemolytic streptococcus in the ear showed the same organism in the throat. The pneumococcus was found in much smaller number and the staphylococcus alone in 22 per cent of the acute cases and alone or combined with other organisms in 70 per cent. The question of contamination enters in here very largely. The diphtheroid bacillus was infrequent in primary culture but played a prominent rôle as secondary invader, being found in 56 per cent of the cases after one to three weeks. Valentine's final conclusion was that the acute cases are due most frequently to the beta hemolytic streptococcus, the staphylococcus, the pneumococcus, and the green streptococcus, and the chronic cases to the staphylococcus, bacillus pyocyaneus, bacillus proteus, bacillus coli and the diphtheroid bacillus. Bacteriology alone does not account for the development of chronicity.

In the study of acute aural infections certain organisms assume peculiar importance. Hemolytic streptococcus is prone to cause a fulminating type of mastoiditis and intracranial complications; hence studies on blood agar plates should always be made. The streptococcus mucosus capsulatus, now generally recognized as pneumococcus type III, is another organism which is characterized by its insidious and extensive invasion of bone tissue and subsequent sudden onset of intracranial complications. This organism is easily recognized in bacteriologic studies by its bile solubility, and by its capsule, and any ear lesion due to it should be watched very carefully for early operative indications.

There are numerous case reports of diphtheritic otitis media, and Drury<sup>60</sup> has presented a very thorough study of this particular infection. Kerrison states that 5 per cent of all cases of diphtheria develop some type of acute otitis media; Duel puts the figure at 10. Only a small percentage of these are true pri-

mary diphtheria, but many of them are due to diphtheria associated with other organisms. A diphtheritic otitis media is occasionally seen in a carrier. The infection occurs through the eustachian tube in cases of pharyngeal diphtheria; Drury feels that the organism probably comes through the blood stream rather than from the pharynx. Otitis media due to a pure infection of diphtheria bacilli is characterized by watery discharge, sodden appearance of the drum, and the possible presence of membrane about the perforation; such findings would suggest the advisability of culturing the discharge on the usual Löffler medium and treatment with antitoxin.

Tuberculosis of the middle ear is a disease the diagnosis of which may be made by bacteriologic or pathologic methods. The tubercle bacillus is recovered with difficulty from the aural discharge, particularly in the chronic cases, which often show an acid fast bacillus which must be carefully differentiated by proper staining reactions. The organism may be found in the discharge from acute cases. An attempt to establish the diagnosis by bacteriologic methods involves the collection of the secretion and its examination either in smear preparation or by injection into guinea pigs. The material for smear preparation should be treated with antiformin and centrifuged, while that which is to be used for animal inoculation should not, of course, be treated with antiformin. Wingrave<sup>61</sup> reported finding bacilli in either discharge or curettage material in 16 per cent of the cases of chronic tuberculosis and in 87 per cent of the acute cases. Such studies should be made in cases presenting multiple perforations of the drum, or in long standing cases which have resisted all therapy and in which bacteriologic studies have given no clue.

One should bear in mind the possibility of finding Vincent's spirochete and the bacillus fusiformis in suppurations of the middle ear. Pilot and Pearlman<sup>62</sup> found these organisms in the foul discharge of chronic, suppurative otitis media, associated usually with streptococcus, pneumococcus or diphtheroid bacillus; it was not found in the normal ear canal, in acute infections, or in nonfetid discharge. The organism comes through the eustachian tube.

Otomycosis is another aural infection, the diagnosis of which is established by bacteriologic methods. Chisolm and Sutton<sup>63</sup>

feel that it is more frequent than is commonly suspected. The fungi belong to two families, the mucoraceæ and aspergillaceæ, seventeen species of which are pathologic for man; ten of these have been reported in various ear conditions, the most common being the *aspergillus fumigatus*. These organisms are probably never primary invaders but occur secondarily in other pathologic conditions; they are found particularly when maceration of the skin has occurred as a result of trauma or prolonged soaking in water. The mycelium may occur not only in the external canal, but also in the middle ear itself. If the infection remains superficial it may cause only slight itching, a feeling of fullness, and impairment of hearing; if the deeper tissues are invaded there may be intense itching, with swelling of the canal, tenderness and dull pain. If the middle ear is involved there is rather profuse discharge of mucus. The infection, particularly in the dry cases, can be recognized by the fine brown, yellow or almost white flakes which present in the external canal. The diagnosis is made by finding mycelia or spores in the slides or in cultures in Sabouraud's agar or Raulin's liquid.

Bacteriologic studies of the mastoid at operation indicate the responsible organism in a large percentage of cases, and when an intracranial complication is present will frequently reveal the organism which is responsible. In acute cases some type of hemolytic streptococcus is usually found. In a series of thirty-five cases 48.3 per cent showed this organism. Such cultures should always be taken whether intracranial complication is present or not.

Among nasal conditions which may be diagnosed by bacteriologic methods are Vincent's infection, diphtheria and primary syphilis. Nasal cultures in pharyngeal diphtheria should be a matter of routine in convalescence in order to rule out carriers. Diphtheria will always suggest itself when one sees in the nose a white, adherent membrane, accompanied by profuse, watery discharge and marked nasal occlusion; such membrane may be seen in scarlet fever and severe streptococci infections, and it is quite essential to rule out its specific character. The fusiform bacillus and the spirochete of Vincent have been reported in severe nasal lesions, particularly in small children, and smear examination should always be made when there is infection

of unknown etiology, particularly if accompanied by profuse, bloody discharge.

Dark field examination for the spirochete pallida should be made in all lesions about the nose when there is any possibility of the condition being syphilitic. Chancre of the septum is a comparatively rare occurrence, but occasional cases are reported and demonstration of the specific organism will allow treatment to be stated when it is of greatest value.

The bacteriology of the throat, like that of the ear, has been the subject of many elaborate researches, most of which have concerned the hemolytic streptococcus. Pilot and Davis<sup>94</sup> found this organism in the crypts of 95 per cent of excised tonsils, on the surface of 61 per cent of such tonsils, and in 43 per cent of nasopharynges. Bloomfield<sup>95</sup> found in the normal throat a nonhemolytic streptococcus, a gram positive coccus, and the influenza bacillus and in lesser numbers pneumococcus type IV, the diphtheroid bacillus and the staphylococcus. He found that the beta hemolytic streptococcus is not a normal inhabitant of the throat and that its presence indicates a focus of infection; it was present in 9.5 per cent of the tonsillectomized cases and in 41 per cent of patients with tonsils. It is particularly located in the bottom of the crypts.

It is academic to speak of the diagnosis of diphtheria by bacteriologic methods, and yet there are one or two points which might be mentioned. In suspicious cases one should not be satisfied with one negative culture but should continue taking them daily until the case either clears up or the diagnosis is established. Here, as in other laboratory methods, a negative result does not rule out the condition suspected. Cases have been reported in which anti-toxin was withheld on account of negative cultures only to be given too late after positive cultures had been obtained. The common practice of smearing the swab over the surface of the membrane is not the best way of taking the culture. If possible the membrane should be lifted up and material obtained from its edge, or preferably under surface, where the organisms are most apt to be found. Many bacteriologists will not make an absolute diagnosis of diphtheria from the smear preparation but prefer to wait for an eight or ten hour culture.



Infection with Vincent's spirochete and fusiform bacillus is common and its diagnosis well understood. The organisms are found about the teeth and tonsils in poor hygienic condition and are frequently responsible for severe gingivitis and tonsillitis. The presence of a few organisms in the smear is not sufficient evidence for concluding that they are causing the pathology present; they should predominate to permit this conclusion.

Pilot and Brams<sup>66</sup> found the fusiform bacillus in 82 per cent of extirpated tonsils and 32 per cent of extirpated adenoids and the spirochete of Vincent in 25 per cent of such tonsils and 2 per cent of such adenoids.

The dark field is important in the diagnosis of suspicious lesions in the mouth and pharynx, particularly the primary sore and the mucous patch; it should always be done when there is any questionable ulcer in these areas.

Actinomycosis is a disease which is peculiarly apt to be found about the head and neck. New and Figg<sup>67</sup> report a series of 157 cases, 107 of which occurred in these areas, and state that the cervicofacial group makes up the majority of these infections. Actinomycosis of the tongue is reported in increasing number and in any obscure lesion of the tongue, glands of the neck, skin or subcutaneous tissue of the cervicofacial region in which malignancy, Hodgkin's or phlegmon is suspected, watch should be kept for the ray fungus and sulphur granules of actinomycosis. These are the macroscopic yellowish white bodies seen in the discharge from actinomycotic cavities and should always be looked for, particularly in freshly opened pockets and deep crypts. Diagnosis is made by crushing a suspected granule between slides and examining for the characteristic mycelia and spores of actinomycosis. The organism is grown culturally but it is not a simple matter. If mixed infection is present the fungus may not be found in the discharge, but must be looked for in the tissues themselves.

#### PATHOLOGY.

Pathologic studies have but a limited use in otology inasmuch as malignant disease of the ear is not common. In a case with polyp or granulation tissue which bleeds freely, recurs promptly or has any feeling on palpation suggestive of malignancy,

biopsy should be done. It may also be done on granulation tissue from chronic suppurative otitis media when the exact etiology is unknown and tuberculosis is suspected; a small bit of tissue may be punched out and examined for tubercle bacilli and giant cells. Wingrave<sup>91</sup> states that chronic tuberculosis is characterized by the finding in curettage material of giant cells but rarely tubercle bacilli, and that acute tuberculosis more frequently shows tubercle bacilli and less often giant cells. One must be careful to differentiate the true giant cell of tuberculosis from the large mononuclear cell frequently seen in non-specific bone granulomata.

In rhinology very much the same general indications for biopsy hold. Whenever a case presents in which the tissue has any suggestion of malignancy, either in free bleeding, early recurrence, or in the hard consistency of cancer, pathologic sections should be made; frozen sections during operation are occasionally desirable when one is desirous of knowing whether the tissue under immediate consideration is malignant or not.

Three pathologic processes occur in the larynx which are often difficult of differentiation, namely, tuberculosis, syphilis and cancer. The question of whether or not biopsy should be done in suspicious lesions of the larynx has long been the subject of spirited discussion. Some maintain that biopsy should not be practiced because of the danger of causing metastasis, others that it should be done only when all other means of diagnosis have been exhausted and the nature of the lesion is still in doubt, while others maintain that biopsy, being a harmless procedure, should be used freely in every suspicious lesion of the larynx, because there are many cases in which the diagnosis cannot be established without its aid. All agree that if tissue is taken it should be done with the least possible trauma, that crushing and bruising manipulations should be avoided, that a sharp punch forceps or cutting instrument should be used, and that the procedure should not be carried out unless permission has been obtained for immediate operation in case malignancy is diagnosed. The question is not settled; the solution apparently depends on the experience of the individual operator and his ability to diagnose malignancy without the microscope.

Actinomycosis is another lesion that may be diagnosed by biopsy, even in the absence of sulphur granules, and the possibility should always be borne in mind in obscure lesions about the head. Jackson<sup>68</sup> has reported a case of blastomycosis of the larynx in which the diagnosis was made by biopsy and culture.

The usefulness of biopsy in general can be stated by saying that whenever we have a tumor, granulation or ulcer of unknown character which is surgically accessible and from which tissue can be removed without hazard to the patient, such study should be made. One criticism of the use of biopsy has been the fact that it is sometimes difficult to be sure the specimen has been taken from the true tumor and not from harmless granulation. Tissue should be taken from the edge and from the center of the growth; even under such conditions first specimens are occasionally reported negative and later ones positive. The explanation is, of course, that the biopsy missed the tumor the first time and found it the next. A second difficulty is the varying reports made on the same slide. Despite these shortcomings the procedure is well recognized and widely practiced.

MILLER CLINIC, HAMM BLDG.

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CHARLES W. RICHARDSON



## XXII.

CHARLES WILLIAMSON RICHARDSON, M. D.

1861--1929.

WASHINGTON, D. C.

Charles Williamson Richardson of Washington, D. C., died in Boston, August 25, aged 68, following an operation. Dr. Richardson was born August 22, 1861, in Washington, D. C. He received his medical degree from George Washington University, at that time known as Columbian University, in 1884. He received the same degree from the University of Pennsylvania in that year. These universities later conferred the honorary degree D. Sc. on him, the University of Pennsylvania in 1927 and George Washington University in 1921.

At the time of his death, Dr. Richardson was a member of the Board of Trustees of the American Medical Association and Professor Emeritus of Laryngology and Otology in George Washington University. He had filled the active professorship in this department from 1891 until 1924.

Dr. Richardson was a frequent contributor to the literature of otolaryngology and had a part in the preparation of several textbooks. He was a Colonel in the Medical Reserve Corps, having served during the Great War as director of the section of defects of hearing and speech in the surgeon-general's office. He was active in many scientific societies, among them the following: fellow and past-president of the American Laryngological Association; fellow and past-president of the American Otological Society; fellow and past-president of the American Laryngological, Rhinological and Otological Society; and a member of the American Climatological and Clinical Society and president from 1921 to 1922. He was also a fellow of the American College of Surgeons; fellow of the Royal Society of Medicine in London; member of Washington Academy of Science and Associated Societies; fellow of the Academy of Social Science; member of the International Tuberculosis Association and of the American Association to Promote the Teaching of Speech to the Deaf.

### XXIII.

#### FIVE LECTURES ON THE PHYSIOLOGY OF THE EAR.\*

(CONCLUDED.)

By W. J. McNALLY, M. D.,

MONTREAL.

#### LECTURE 3.

#### PHYSIOLOGY OF THE UTRICLE AND ITS ASSO- CIATION WITH POSTURE.

The utricle is part of the vestibular labyrinth and is one of the so-called otoliths. It is situated in the pars superior of the labyrinth. In fact, the utricular macula rests on the dividing membrane of deBurlet. The macula lies in the horizontal plane on the floor of the utricular chamber. Opening into this chamber above are all three semicircular canals, the utricular chamber completing the circle of each canal to allow for a flow of endolymph through the canal. The utricle is connected below through the utriculosaccular duct to the saccule and ductus endolymphaticus which have already been studied.

Construction of the Macula.—The utricular macula consists of a disc or cup shaped area of specialized epithelium, which supports the nervous hair cells. Resting on the hair cells is a membrane of greater specific gravity, the otolithic membrane, the weight of which acts as a direct stimulus to the hair cells. The nerve from this specialized area is called the utricular nerve and it joins the nerves from the ampullæ of the anterior vertical and horizontal semicircular canals.

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**General Function.**—In discussing the general function of the labyrinth, it was pointed out that the semicircular canals are stimulated by a sudden movement or change of movement of the head, whereas the otolithic mechanism is affected by a change of position affecting the center of gravity of the animal. It is a gravity phenomenon. Breuer (1875) was the first to make this definite differentiation of the labyrinthine reflexes. When the position of the head is changed, there is a necessary readjustment of the limbs and body. This readjustment is maintained, for the most part, by the intact otolithic mechanism. In the previous lecture, it was shown that the saccule has not been proved to have any vestibular or equilibrical function, so that the utricle must give rise to the reflexes previously ascribed to both the utricle and saccule.

**Mechanism of Utricular Function.**—Much speculation has taken place as to the actual mechanism of the utricle. It is generally admitted that it is the overlying membrane which stimulates the hair cells of the macula, and it is further admitted that owing to the greater specific gravity of the otolithic membrane it is affected by any change of position of the head out of the horizontal plane—that is, any change which causes a shifting of its center of gravity. The main difference of opinion exists about the manner in which the otolithic membrane stimulates the hair cells. The older idea was that it was the pressure of the otolithic membrane on the hair cells. With any change of position of the head, the otolithic membrane presses on a different part of the macula, and, therefore, a different set of reflexes is elicited. The main exponent of this idea today is Quix. He believes that the pressure of the otolithic membrane on the macula is the maximal stimulus, and that the resulting reflex is an increased tone in the flexor muscles of the limbs.

The newer theory propounded by Magnus and deKleijn is that it is the pull of the otolithic membrane on the macula that is the maximum stimulus to the hair cells, and that the resulting reflex is an increase in the extensor tone of the limbs. They believe that pressure of the membrane on the macula is a minimum stimulus, which causes an increase in the tone of the flexor muscles of the limbs. This last theory arose from a study of the topography of the otolithic maculae when certain

increased  
tonus in  
extensor muscles

definite reflexes are most marked. They found that when a decerebrate cat with intact labyrinths is placed on its back, the head being tilted back so that the line of the snout makes an angle of  $45^\circ$  above the horizontal plane, there is a very strong extension of all four limbs. To avoid turning of the head, neck and thorax, these were often fixed in a plaster jacket, care being taken to allow free movement of the limbs. When this same preparation or animal is placed in the ordinary standing position, the head bent forward, so that the line of the mouth forms an angle of  $45^\circ$  below the horizontal plane, extension of the limbs is at a minimum. The limbs, in fact, become flexed. If both labyrinths are removed from a decerebrate animal, the strong extension of the limbs no longer occurs when the animal is placed on its back. In fact, when the head is fixed to the rest of the body by a plaster jacket, any change in the position of the animal does not affect the tone of the limbs. It was decided, therefore, that this change of tone in the limb muscles in the decerebrate preparation arises from the intact labyrinths. From a study of the position of the different otoliths when the extension of the limbs is most marked, it was discovered that the utricle maculae are in the horizontal plane with the otolithic membranes hanging from the maculae—that is, they are upside down. The saccular maculae and otoliths are in no characteristic position. It was, therefore, argued that this maximum increase of tone in the extensor muscles of the limbs arises from the utricular macula when the otolithic membrane is hanging from the macula, and that this hanging or pulling of the otolithic membrane on the macula is the maximum stimulus to the macula. When the animal is placed in the normal standing posture and the extensor tone of the limbs is at a minimum—that is, the limbs are flexed, the otolithic membranes, of course, are pressing on the macula, and they, therefore, decided that this is the minimum stimulus to the utricular macula.

If only one labyrinth is removed and the decerebrate animal is again placed on its back, the head being held symmetrical to the rest of the body by a plaster jacket and tilted back so that the line of the mouth forms an angle of  $45^\circ$  above the horizontal plane, there is a maximum increase of tone in the extensor muscles of the limbs just as when both labyrinths

are still present. They decided, therefore, that each labyrinth supplies the increased extensor tone to the limb muscles of both sides of the body—that is, to all four limbs. They further decided that the utricular macula influences the tone of the neck muscles, the maximum and minimum position being the same as for the limbs, but that each utricle is only connected with the muscles of one side of the neck, and that is the opposite side. For this reason, following a unilateral labyrinthectomy the head is turned to the operated or delabyrinthized side. From a careful analysis of these two theories it will be seen that both authors are agreed upon the reflex effect upon the limbs from either pressure or pulling of the otolithic membrane. They differ only in deciding which effect shall be considered maximal. For example, if the otolithic membrane is pressing on the macula, both authors agree that the effect upon the muscles of the limbs is a uniform increase of tone in the flexor muscles and a decrease of tone in the extensor muscles of all four limbs. Quix would call this a maximum utricular response, but Magnus would call this a minimum utricular response. On the other hand, if the otolithic membrane is hanging from the macula, both authors agree that the effect upon the limb muscles is a uniform increase in the tone of the extensor muscles of all four limbs and a decrease in the tone of the flexor muscles of all four limbs. Quix would consider this a minimum utricular response, whereas Magnus would consider this as a maximum utricular response.

**Tonic Labyrinthine Reflexes on Body Musculature.**—The reflexes which result when an animal is placed on its back, its head being tilted back so that the line of the snout forms an angle of  $45^\circ$  above the horizontal plane—that is, the increased tone in the extensor muscles of all four limbs and the increased tone in the muscles of the opposite side of the neck form the basis of what Magnus calls the tonic labyrinthine reflexes on the body musculature. As has been mentioned, they disappear after bilateral labyrinthectomy. They were definitely localized to the otolithic mechanism by Magnus and deKleijn. In subjecting guinea pigs to rapid centrifuging by the method of Wittmaack, the otolithic membranes are displaced from the maculae, the semicircular canal mechanism being left intact.

When these animals have recovered and are examined in the supine position, which should bring about the reflex of maximum extensor tone in the limbs, it was found that this increased extensor tone is not present, yet all reflexes arising from the semicircular canals are normally present. These experiments definitely localized this tonic labyrinthine reflex on the muscles of the limbs to the otolithic mechanism. The localization of these tonic labyrinthine reflexes on the body musculature to either the utricle or the saccule was arrived at by a study of the characteristic position or topography of each of these maculae when the tonic reflex was maximum. As was mentioned before, these reflexes are maximum when the animal is on its back with the head tilted back, so that the line of the snout makes an angle of  $45^\circ$  above the horizontal plane. In this position, both utricular maculae are in the horizontal plane, whereas the saccular maculae are practically in the vertical plane and in no characteristic position for stimulation of a macula by either pressure or pulling of its otolithic membrane. By this topographical argument, it was decided that the tonic labyrinthine reflexes on the body musculature arise in the utricular maculae, and that each utricle affected the tone of the muscles of all four limbs, but of one side of the neck, and that the opposite side.

Attitudes.—Magnus has shown that it is possible to impress upon the whole body different adapted attitudes by changing only the position of the head. This implies two different factors:

1. A change in the position of the head in relation to the body by which receptors in the muscles of the neck are stimulated. The reflexes which result are called neck reflexes.

2. A change of orientation of the head in space which brings the labyrinths into play. These are the labyrinthine reflexes.

The first set of reflexes—the neck reflexes—can be eliminated by immobilizing the head, neck and thorax in a plaster jacket. This was done in the experiments just described to study the tonic labyrinthine reflexes on the body musculature in the decerebrate cat.

Neck Reflexes on Limbs.—It was shown by Magnus and deKleijn that the first group of reflexes—neck reflexes—can

best be studied in a delabyrinthized animal, for instance, a delabyrinthized rabbit. When such an animal is placed in a plaster jacket to immobilize the head, neck and thorax, it can be put in any position without causing change of tone in the body musculature. However, if the head of a delabyrinthized animal is rotated to one side or the other, there is an immediate change of tone in the limb muscles, and it is usually asymmetrical. The limbs on the side to which the snout is turned are extended and abducted, and the limbs of the opposite side are adducted and flexed. Again, if the head of such an animal is elevated, the front legs are extended and the hind legs are flexed, or, if the head is depressed, the front legs are flexed and the hind legs are extended. These reflexes disappear after section of the dorsal cervical roots. Magnus and deKleijn have, therefore, shown that these different attitudes are "chain reflexes," which take origin in receptors situated in the muscles of the neck, so that when these muscles contract they affect the tone of the muscles of the lower segment of the body—the thorax—where in turn other "chain reflexes" are set up to act upon the muscles of a segment still lower. These tonic neck reflexes are responsible for many of the attitudes assumed by normal animals. For instance, when a dog sits at the bottom of a tree watching a cat up in the branches.

Neck Reflexes on the Eyes.—Bárány noticed that if the head of a normal rabbit is fixed so as to eliminate labyrinthine reflexes, it is possible by simply moving the body to cause a change in the position of the eyes. If the body is moved to one side or the other or upwards or downwards, or if the body is rotated on its longitudinal axis, there is a slight but definite movement of the eyes in the opposite direction. These are compensatory eye movements from tonic neck reflexes. Magnus and deKleijn made a more thorough analysis of these eye movements by studying them in the delabyrinthized rabbit, the head being fixed by a Czermak clamp. If the body is moved over to the right, there is a conjugate deviation of the eyes to the left. Again, if the body is rotated down and to the right, the right eye moves up and the left eye down. These eye movements disappear after section of the dorsal cervical roots. They are, therefore, tonic neck reflexes on the eye muscles. They tend to help the eyes to retain their original field of vision,

even when the head is moving. From definite measurements it was found that these tonic neck reflexes can produce in the rabbit an average compensatory eye movement of about  $30^\circ$ .

**Labyrinthine Compensatory Eye Positions.**—It will be remembered that in discussing the physiology of the saccule we mentioned compensatory eye positions which disappeared after the removal of the otolithic membranes by centrifuging. These reflexes are best studied in animals with laterally placed eyes, such as the rabbit or the guinea pig. When the visual fields overlap, as in the cat, dog or monkey, narcosis is necessary to study these reflexes. These reflexes are best seen when the whole animal is fixed immovably to a board, the head being fixed by a Czermak clamp. If the whole animal is rotated, for example, down and to the right, the right eye moves up and the left eye down. This compensatory eye movement seems to be an attempt to retain the original field of vision. However, Magnus and deKleijn found that if actual measurements are made when the body is rotated through an angle of  $90^\circ$ , the eyes move back only through an angle of  $70^\circ$ , so that it is not a complete compensation by the eye but only an attempt at compensation. These reflexes are not present in the delabyrinthized animal or in an animal after the otolithic membranes have been detached by centrifuging. These reflexes are still present after destruction of the saccular macula and otolith in the rabbit (Versteegh). It, therefore, becomes evident that these compensatory eye movements and positions, which are brought about by a movement of the whole animal and which are abolished by bilateral labyrinthectomy, really arise in the utricular maculae. However, as has just been pointed out, these utricular compensatory eye reflexes do not actually bring about complete compensation, but, as computed by Magnus and deKleijn, only about 80 per cent. It will be remembered, however, that the tonic neck reflexes on the eye muscles cause a compensatory eye movement of about 20 per cent. Furthermore, when a normal rabbit moves its head, he is moving it in relation to space—otolithic stimulation, and he is moving it in relation to the rest of his body—neck reflexes, so there would be acting on the eye muscles a combination of these two reflex compensatory eye movements, which together would bring about perfect compensation. In



the higher animals, with overlapping fields of vision, these reflexes just described are subordinate to ocular righting reflexes, but these labyrinthine and tonic neck compensatory eye reflexes are still present, though much less evident. In man an attempt has been made to measure these reflexes. It was thought that the rotatory compensatory eye reflexes originated in the saccule, and by measuring these compensatory eye movements it was hoped that some knowledge of the state of functioning of the saccule could be ascertained. The methods used are extremely complex and have never received very general application—whether they will still be used when it is generally recognized that these reflexes do not arise from the saccule and do arise from the utricle, I do not know. If the method could be simplified the interpretation of the results would at present be less misleading, if it is realized that these reflexes originate in the utricle.

**Righting Reflexes.**—In discussing the saccule, head righting reflexes were referred to. Righting reflexes were defined as those by which an animal from every abnormal position in space is able to restore itself to a normal position. There are five groups of reflexes cooperating in this:

(a) The labyrinthine righting reflexes acting on the head. Breuer was the first to localize this reflex to the labyrinth, because it disappeared after bilateral labyrinthectomy in the guinea pig. This reflex may be shown by grasping the normal rabbit by the pelvis and holding it in the air. No matter in what position the pelvis is held the head is always maintained in the normal position in space. When the body or pelvis is held to one side and the head is righted or maintained in its normal position in space, this reflex is called by Magnus an asymmetrical righting reflex. This reflex was previously ascribed to the saccule. However, as was shown in the last lecture, section of the saccular nerve in the frog or destruction of the saccular macula in the dogfish or the rabbit does not interfere with any of the righting reflexes. When the pelvis is held so that the animal should really be lying on its back, the head will still be maintained in the normal erect position. There is no asymmetry about this position of the animal, because the torsion of the body is about a horizontal axis. This reflex is called the symmetrical labyrinthine head

righting reflex. Both these reflexes are absent in the delabyrinthized rabbit or guinea pig, and they are absent after the otolithic membranes have been detached by centrifuging. They are, therefore, otolithic in origin, and as they are not affected by denervation or destruction of the saccule it follows that they arise from the intact utricles. They were absent in the rabbits in which Versteegh succeeded in destroying the utricular nerve, leaving the remainder of the labyrinth intact.

(b) The Body Righting Reflexes on the Head.—If a delabyrinthized thalamus rabbit (one with the cerebrum removed) is held in the air on its side, it remains perfectly quiet. However, if it is lowered to the table in the lateral position, it immediately brings its head into the normal attitude. This reflex arises from the asymmetrical stimulation of the skin on the lower surface of the body. This can be proven by laying a heavy board on the upper surface of the animal's body when the head returns to the lateral position.

(c) Neck Righting Reflexes on the Body.—When the body is held in an abnormal position, as when the rabbit is held by the pelvis in the air, the head is righted because of the above reflexes, labyrinthine and body righting reflexes on the head, but furthermore, the upper part of the thorax, etc., is also righted as far as possible. If the dorsal cervical roots are sectioned, the head is righted but not the thorax, etc. The head being righted causes a change in the tone of the neck muscles, which sets up a righting reflex in the thorax, etc., which brings the rest of the body into line with the head.

(d) The Body Righting Reflex on the Body.—If a normal rabbit is held in the air so that its head is held in the lateral position, the animal lies quietly. If the animal is gradually lowered to the table, still in the lateral position, but with the head firmly grasped so that it cannot move, the moment the body touches the table, the animal immediately scrambles to its feet, in spite of the fact that the head is still held in the lateral position. This reflex that is strong enough to overcome the neck righting reflexes, which tend to keep the body in symmetry with the head, can be eliminated by applying a heavy board to the upper surface of the body, showing that this reflex originates from asymmetrical stimulation along the lower side of the body.

(e) The Optical Righting Reflexes.—This last righting reflex is not present in the rabbit or the guinea pig and only present in higher mammals with an intact cerebrum—cat, dog, etc. (the other four reflexes are all present in the thalamus animal).

If a labyrinthized cat with cerebrum intact is held in the air by the pelvis, it keeps its head in the normal position in space. However, if the animal is blindfolded, it no longer rights the head, the head being in symmetry with the rest of the body. It now acts like a labyrinthized thalamus cat or a labyrinthized rabbit.

Head Righting Reflex in a Frog.—In order to show head righting reflexes of a frog—that is, its response to gravity or change of position, the frog may be placed on a board and tilted out of the horizontal plane. Whether it is tilted backwards or forwards or to either side, in each case there is readjustment of the limbs and body so that the head is maintained in its horizontal position. This is a response to slow tilting out of the horizontal plane. This reflex is still present in a frog if the ampullæ of all six semicircular canals have been destroyed, and after section of the nerves to both saccular maculæ. However, it disappears after the removal of both labyrinths. It is, therefore, a labyrinthine righting reflex, most probably arising from the utricular macula and acting upon the head. Because the righting of the head to slow tilting disappears after bilateral labyrinthectomy in the frog, it would seem that the body and ocular righting reflexes on the head present in the mammal cannot be very strong in the frog.

Centrifugal Force Acting Upon a Frog.—Another very interesting righting reflex has been demonstrated by Tait and McNally in the frog. It is the same righting reflex as has just been discussed, but it is in response to a different stimulus than gravity. It is the righting reflex in response to centrifugal force. Centrifugal force is that force which is acting on a body when it is being rotated rapidly and which tends to throw it away from the center. For example, the pull exerted by a stone, which is tied to a string and swung over the head. When there are two such forces acting—gravity and centrifugal force—the animal reacts to the resultant of the two forces.

There is a readjustment of the animal's body so that the line of force passes through the center of the body and at right angles to the utricular macula. Hence the animal which is being rotated or which is running around a corner leans in towards the center. For example, a race horse making a sharp curve or a man on a bicycle.

Kreidl found that when fish are rotated, they lean in to the center, reacting to the resultant of gravity and centrifugal force. Tait and McNally showed that when a frog is rotated on a turntable, side to the center (subjected to centrifugal force) the speed of rotation being increased so slowly as not to stimulate the semicircular canals (angular acceleration being kept below the threshold of stimulation), the frog leans in toward the center to protect itself from being thrown off the table. The lean becomes more marked as the speed of rotation is raised, because the amount of centrifugal force is also increasing. If all six semicircular canals were removed or if both saccular nerves were sectioned, the animal still reacts normally to centrifugal force, but a frog in which both labyrinths have been destroyed does not lean to the center when rotated on the table, and, like an inanimate object, is soon thrown off. These experiments, therefore, show that an intact utricle is necessary for a normal righting reflex in response to centrifugal force, and they show, furthermore, that the utricle is affected by any continuous force, which acts upon the animal in a direction other than that of the pull of gravity, such as centrifugal force.

In considering these righting reflexes, it might be pointed out that there are three sets of reflexes righting the head in relation to space—otolithic, tactile and ocular reflexes, and there are two sets of reflexes righting the body—a deep or proprioceptive reflex from the neck muscles and a tactile reflex from the body surface.

Clinical Applications of Righting Reflexes.—These reflexes have an important clinical bearing because in carrying out Rhomberg's test we are more or less looking for these reflexes; a person depends upon eye, labyrinthine and tactile sensations (including kinesthetic sensation) for maintaining normal posture. In tabes, the tactile righting reflex is absent.

In a diseased labyrinth the utricular righting reflex is gone. As a matter of clinical experience in doing Rhomberg's test, the patient is asked to close his eyes. This eliminates the ocular righting reflex. If one of the other two righting reflexes, the tactile or otolithic, is absent from disease, then the patient sways or falls, because one set of righting reflexes is not sufficient to maintain balance.

**Unilateral Labyrinthectomy.**—Following a unilateral labyrinthectomy there is a marked turning of the head to the side operated upon. This is due to the tonic labyrinthine effect on the muscles of the neck from the remaining intact labyrinth. There is a loss of compensatory movements following a turning of the head to the side operated upon, so that if there is a right-sided labyrinthectomy, contrary to what happens in the normal animal, the right eye is turned down and the left eye is turned up. There is a loss of labyrinthine head righting reflexes on turning the head to the side operated upon. There is also an increased tone in the extensor and abductor muscles of the homolateral limbs, and increased tone in the flexor and abductor muscles of the contralateral limbs. This asymmetry of muscle tone in the limbs was at first considered to be a true labyrinthine reflex. However, it was shown by Magnus and deKleijn that the asymmetry of the limbs is really a neck reflex. If, following a unilateral labyrinthectomy, the turning of the head is corrected so that the head is brought into symmetry with the rest of the body, the asymmetry of the limbs disappears, but the asymmetry of tone in the neck muscles and also the loss of labyrinthine righting reflexes and compensatory eye movements to turning of the head to the side operated upon persist.

**Turning of the Head Following Labyrinthectomy.**—Magnus and deKleijn showed that this turning of the head to the side operated upon was due to a reflex influence from the opposite intact labyrinth. This was proved by the fact that the turning of the head persisted even after cocain was injected into the destroyed labyrinth. The cocain would anesthetize and eliminate any irritative phenomenon which might send out impulses from the destroyed labyrinth.

While in Magnus' laboratory, I was asked to determine what cervical nerves were concerned in this turning of the head fol-

lowing a unilateral labyrinthectomy. The rabbit was the experimental animal used. After a unilateral labyrinthectomy the cervical nerves of one and both sides in different combinations were sectioned in a large number of animals. It was concluded that the impulses from the intact labyrinth crossed to the cervical nerves of the opposite side—that is, the first five cervical nerves and the accessory nerve—and that the crossing does not take place in the medulla and probably does in the cord. It was further concluded that the nerves in the intact side oppose and may partly mask the action of the nerves on the side operated upon.

McNally and Tait (1925) showed that after removal of all three semicircular canals of one labyrinth in the frog, there was no turning of the head, the posture of the animal remained normal when at rest. They also showed that section of one saccular nerve did not produce any turning of the head. These experiments seem to indicate that this increased tone in the muscles of the side of the neck operated upon following a unilateral labyrinthectomy is brought about by the intact utricle. This conclusion was further borne out by the fact that while operating on the horizontal or anterior vertical canal in the frog, great care has to be exerted not to injure the utricle, which is close by. If the utricle is injured, there always follows a turning of the head to the injured side. We have been so far unable to carry out a lesion of the utricle only in the frog without injuring the other labyrinthine structures.

Destruction of Utricular Nerve.—Versteegh (1928) has succeeded in destroying the nerve to the utricular macula in the rabbit without interfering with any of the other structures in the vestibular labyrinth. Following this operation the animal has lost all the reflexes formerly ascribed to both the utricle and saccule of one side—the reflexes responding to changes of position, whereas all reflexes in response to sudden movement are still present. The animal has lost compensatory eye movements and labyrinthine head righting reflexes in response to turning of the head to the side operated upon. The head is rotated to the side operated upon—that is, the animal adopts the attitude which usually follows unilateral labyrinthectomy. It differs from the delabyrinthized animal,

because the turning and caloric reactions and progression reactions are quite normal on both sides. By this experimental extirpation of the saccule and utricle individually in the rabbit, Versteegh has crystallized the evidence pointing to the fact that the utricle is the seat of the labyrinthine head righting reflexes, labyrinthine compensatory eye movements and positions and the labyrinthine tonic influence on the body and neck musculature. It is this labyrinthine tonic effect on the neck muscles of the opposite side which causes the turning of the head following a unilateral utricular injury.

**Clinical Significance of Rotated Position of Head.**—This turning of the head following a utricular lesion has an important clinical bearing. Holding of the head to one side or the other is a common symptom in certain brain lesions. It was formerly called the cerebellar posture of the head. Horsley was the first to suggest that the rotation and flexion of the head are usually due to a lesion of the labyrinth rather than to a lesion of the cerebellum. Rademaaker in Magnus' laboratory has removed the cerebellum in a large series of cats, dogs and monkeys. Following the removal of half or the removal of the whole of the cerebellum, the head is held symmetrically in all cases. Brain summarized the most generally accepted clinical significance of such a lesion as follows:

(a) Unilateral otitis interna or an acoustic nerve tumor leads to rotation of the head to the affected side.

(b) Lesions of the pons and midbrain have been found to lead to rotation of the head to the normal side.

(c) Lesions confined to the cerebellum do not lead to the rotated posture of the head. If there is rotation of the head in cerebellar disease, it is suggested that if the turning is to the side of the lesion, it is due to pressure on the homolateral eighth nerve. If it is due to the side opposite the lesion, which occurs in more advanced cases, it is due to compression of the pons and medulla on the affected side.

We may summarize by saying that rotation and flexion of the head may follow disease of the labyrinth, eighth nerve, pons, midbrain or forebrain. It does not follow disease confined to the cerebellum, and is most probably actually due to

a lesion of the utricle or its intracranial connections. It may, however, be due to a unilateral eye muscle paralysis.

#### SUMMARY.

The utricular macula is an organ of static equilibrium. It is affected by any change of position of the head—response to gravity stimulation. It is also affected by any steady force acting upon the animal in a direction other than the pull of gravity, such as centrifugal force.

The utricle influences the tone of the muscles of all four limbs symmetrically. It affects the tone of the muscles of the opposite side of the neck.

The utricle is the seat of origin of the labyrinthine righting reflexes on the head.

It is also the seat of the labyrinthine compensatory eye positions. Associated with the utricle in bringing about this tonic influence on the body musculature and on the eyes, are the neck reflexes on the limbs and eyes.

Associated with the utricle in righting the head are the righting reflexes from the body and from the eyes.

The utricle maintains indefinitely any attitude which has been rapidly assumed from reflexes from the semicircular canals in response to a sudden movement of the head.

#### LECTURE 4.

#### THE PHYSIOLOGY OF THE SEMICIRCULAR CANALS.

As the utricle is the organ of static equilibrium and is affected by change of position, so the semicircular canals are the organs of kinetic equilibrium, and are affected by sudden movement or change of movement. This difference between the canals and the otoliths was first brought out by Breuer.

Anatomy of the Semicircular Canals.—There are three semicircular canals in each ear. They each open into the utricular chamber at both ends of the semicircle, the two vertical canals having a common opening for their nonampullated ends. At one end of each canal is the dilated ampulla containing the crista, which is the actual nerve end organ. Overlying the hair



cells of the crista is the gelatinous cupula, which is floating in the endolymph. The nerves from the three ampullæ unite with the nerves from the otoliths to form the vestibular nerve, which joins with the cochlear nerve in the internal auditory meatus. In the internal auditory meatus, the vestibular branches enter Scarpa's ganglion.

The three semicircular canals of each ear are so arranged at right angles to each other that in each of the three planes of the body there is a canal in that plane in each ear but oppositely directed. For example, there is a canal in the horizontal plane in each ear and they are oppositely directed in the sense that the ampulla of the horizontal canal in the right ear points forward and to the right, whereas the ampulla of the horizontal canal in the left ear points forward and to the left. One anterior vertical canal is in the same plane but oppositely directed to the posterior vertical canal of the other ear. It was Crum Brown who first pointed out this symmetrical arrangement of the ampullæ being placed at the opposite ends of each pair of canals lying in the same plane. Any movement in a given plane when in one direction affects the canal of one side. When the movement is in the opposite direction, it is detected by the opposite canal in the same plane.

The Mechanism of the Canals.—The accepted theory for the mechanism of stimulation of the semicircular canals is that with any sudden movement of the head there is a lag from inertia of the endolymph and cupula in the canal which is lying in the plane of the movement. This movement of the cupula stimulates the hair cells of the crista so that a nerve impulse is sent out to the central nervous system to bring about a readjustment of the eyes, limbs and body to compensate for the new position of the head. This canal response is very rapid but it is not lasting. However, the new position will be maintained by tonic impulses from the more slowly responding utricles which have been stimulated by the change of position of the head. Flourens and DeCyon found that injury to a semicircular canal in the pigeon, rabbit and frog caused striking abnormality in the animal's behavior. There were spontaneous movements of the whole body and of the eyes and head (eye nystagmus and head nystagmus) after an anterior vertical canal was injured. The animal tends to fall

forward in the plane of that canal. If both anterior vertical canals are injured, the animal topples forward heels over head, and throws itself about in confusion. If both posterior vertical canals are injured, the animal falls backwards, head over heels. Some of these results are evanescent, whereas in some cases permanent forced positions result, as they did not accurately differentiate between the lesion of a canal alone and an associated injury of the otolithic mechanism. Furthermore, they injured the canal rather than the ampulla of the canal.

McNally and Tait (1925) found that in the frog the destruction of one or of all six ampullæ of the semicircular canals caused definite abnormality of movement, as was suggested by the experiments of Flourens and DeCyon. When at rest these animals always assumed a normal pose after the immediate operative irritation had subsided. There was no disturbance of posture of the limbs and, therefore, no disturbance of muscle tone. It is only on movement that abnormality is shown after a lesion of the ampulla. Once the effect of the movement has passed, the animal again assumes a normal posture.

Nystagmus.—Eye movements (nystagmus) are among the most important clinical signs of labyrinthine involvement from disease. This is partly due to the fact that eye movements are so obvious or are so easily looked for. As yet there is not complete agreement as to what part of the labyrinth may cause eye nystagmus. Many authors refer to nystagmus resulting from injury or disease of the otoliths. Nystagmus, however, is usually associated with injury or disease of the semicircular canals. In the centrifuge experiments of Magnus and deKleijn whereby the otolithic membranes were thrown off from the maculæ, there was no spontaneous nystagmus once the immediate effect of the centrifuging had passed. This would tend to eliminate the otoliths as a cause of spontaneous nystagmus. Versteegh has destroyed both the saccule and the utricle in the rabbit, and yet there was no spontaneous nystagmus of the eyes or head, and normal nystagmus could still be elicited to turning or caloric stimulation. These experiments, therefore, would seem to eliminate definitely the otoliths as a cause or source of eye nystagmus. When nystagmus is present in the course of labyrinthine disease, it would suggest canal irritation rather than otolithic irritation.

**Flourens' Law.**—Flourens was the first to describe nystagmus following injury to the semicircular canals. In the pigeon he found that injury to a horizontal or vertical semicircular canal produces a nystagmoid movement of the eyes and head in the plane of that canal. Flourens concluded, therefore, that injury to a semicircular canal causes nystagmus of the eyes and head in the plane of the injured canal. This conclusion has become known clinically as Flourens' law—a semicircular canal causes nystagmus in its own plane. The clinical application of Flourens' law has come to mean that stimulation of a horizontal canal causes a horizontal eye nystagmus, whereas stimulation of a vertical canal causes a rotatory nystagmus in the sagittal plane. There is rotatory nystagmus in the sagittal plane to one side or the other. It is not diagonally up or diagonally down in the plane of either vertical canal. The reason of this is because it is almost impossible clinically to stimulate only one vertical canal of the same ear either by turning or by caloric stimulation. The resulting nystagmus, therefore, in each case is from a combined stimulation of both vertical canals of the same side. For instance, in the case of stimulation of the left ear, stimulation of the left anterior vertical canal would tend to produce a nystagmus or rolling movement of the eye downwards and forwards to the left. The left posterior vertical canal would tend to produce a rolling movement of the eyes backwards and upwards to the left. The resultant of these two attempts is a simple rotatory movement of the eye to the left, neither upwards nor downwards. Clinically, therefore, stimulation of the vertical canals of one ear produces a rotatory nystagmus to one side or the other. If it were possible clinically to stimulate only one canal, the nystagmus, as stated by Flourens, would be in the plane of that canal. It would be diagonally upwards or downwards, but from one vertical canal or from the two vertical canals of one ear the nystagmus could never be vertical.

A vertical nystagmus upwards or downwards presupposes the simultaneous action of the two anterior vertical canals, one from each side, or the two posterior vertical canals. For this to occur clinically, therefore, it would mean simultaneous stimulation or irritation of a posterior vertical canal in each ear or of an anterior vertical canal in each ear. However, it

is uncommon to have such a symmetrical clinical lesion situated in each internal ear. The two anterior vertical canals or the two posterior vertical canals may be affected simultaneously by irritation or injury to their intracranial connections, which lie of necessity in close relation in the brain stem. It is conceivable, therefore, that a lesion in the brain stem may affect the vestibular tracts from both ears, and, therefore, affect the nerve fibers from each anterior vertical canal at the same time or from each posterior vertical canal at the same time, and this simultaneous irritation would produce a vertical eye nystagmus either upwards or downwards. It is for this reason that a spontaneous vertical eye nystagmus is considered diagnostic of a lesion in the brain stem, the brain stem being the most probable place to have simultaneous irritation or stimulation of the two anterior vertical or the two posterior vertical canals or their intracranial connections.

Breuer and Ewald found that if a semicircular canal in the pigeon is stimulated by causing a movement of the endolymph in this direction there is a change in the direction of the eye nystagmus. For instance, if the endolymph in the right horizontal canal is pressed towards the ampulla, there is a movement of the eyes to the left, whereas if the endolymph is pressed away from the ampulla there is a movement of the eyes to the right side. They found, however, from so stimulating the different canals that each canal may be stimulated by a flow of endolymph in either direction but that each canal is maximally stimulated in only one direction.

Ewald's First Law.—From these experiments, they concluded that a horizontal canal is maximally stimulated by a movement of the endolymph in the canal towards its ampulla, and that each vertical semicircular canal is maximally stimulated by a movement of the endolymph in the canal away from the ampulla.

This theory of the maximum and minimum stimulation of each canal applies to the pigeon (Ewald), to the rabbit (de-Kleijn) and most probably to the human (Ruttin). After destruction of one labyrinth in man, it is possible to get responses to turning in both directions, and as pointed out by Ruttin, after a long time has elapsed after the destruction of

the labyrinth the response to turning in either direction is about equal. This theory of maximum and minimum stimulation for each canal does not hold throughout the animal kingdom. Maxwell in the dogfish and McNally and Tait in the frog could detect responses from a semicircular canal in only one direction. This can be definitely shown following the destruction of the ampulla of a horizontal canal in the frog. If the frog is placed on a turn-table, during turning to the side operated upon the animal makes no response, but if the turning is stopped suddenly—after rotation—the animal immediately starts turning in the direction in which the table was originally turned. When turned to the normal side, the animal responds by walking around in the opposite direction. If the table is stopped suddenly, the animal stops moving and remains perfectly quiet. There is no response this time to after-turning. It is quite definite, therefore, that in the frog the horizontal semicircular canal is stimulated by a flow of the endolymph in only one direction.

Ewald's Second Law.—From his experiments Ewald further concluded that when a canal, either horizontal or vertical, is maximally stimulated it causes a nystagmus to its own side—that is, the quick component of the nystagmoid movement is directed towards the stimulated ear. If minimally stimulated, the nystagmus is towards the opposite ear.

Direction of Nystagmus.—There is not complete uniformity of opinion as to whether the direction of a nystagmus should be designated in terms of slow movement or quick movement. In the great majority of cases, and more especially by clinicians, the direction of the nystagmus is referred to in terms of the quick movement, so that by a nystagmus to the right is meant a nystagmus with the quick movement towards the right. This difference of opinion is owing to the fact that only the slow component of a nystagmus is truly labyrinthine in origin. It was originally thought that the quick movement was in the nature of a quick recoil and was cerebral in origin. In support of this belief, it was pointed out that the quick phase of nystagmus is absent during narcosis. deKleijn has clarified this whole question greatly. He showed that a normal nystagmus could be elicited when the cerebrum is completely

removed. He further localized the seat of origin of the quick-phase of nystagmus to the brain-stem in the region of the vestibular nuclei, so that even if the quick phase of nystagmus is not definitely labyrinthine it arises in connection with the intracranial terminations of the vestibular or labyrinthine nerves. deKleijn has since shown, however, that there is a definite cerebral factor in nystagmus. For instance, if only half of the cerebrum is removed in a rabbit, nystagmus can still be elicited from each labyrinth, but the quick phase of nystagmus is accentuated towards the side of the lesion. For instance, if the right cerebrum is removed, there is a more exaggerated nystagmus from stimulating the canals of the right ear by hot water, which movement causes a quick nystagmus to the right, than there is from stimulating the right ear with cold water, which produces a quick nystagmus to the left. On the other hand, there is a more exaggerated response from stimulating the canals of the left ear with cold water, which normally produces a quick nystagmus to the right, than there is from stimulating the left ear with warm water, which normally produces a quick nystagmus to the left.

deKleijn has found a clinical application of this fact in a series of brain tumor cases. In a series of cases where there was gross destruction of the frontal or temporal lobes from tumor or abscess, he found after caloric irrigation of both ears that there is an accentuation of the quick phase of the nystagmus to the side of the lesion, as was the case in the rabbit where half the cerebrum has been removed. In some of the clinical cases the lesion was proved either at operation or postmortem. It is hoped that this point may help in the differential diagnosis of lesions of the frontal lobe.

The three laws of Flourens and Ewald form the basis for the interpretation of the nystagmus which results from the clinical tests for detection of labyrinthine disease.

**Caloric Test.**—Bárány found that irrigating the ear of a normal patient with water, the temperature of which is above or below body temperature, causes an eye nystagmus. The explanation commonly given is that the cold or the hot water changes the temperature of the fluid in the vestibule so that convection currents are set up in the canals. Remembering

the physical principle that a cold liquid falls to the bottom of the container and that hot liquid rises to the top, it is possible to apply this principle to the fluids in the labyrinth, and to predict the resulting response on the eyes. For instance, when the head is erect, the vertical canals are in the vertical plane. If the fluid in the vestibule is cooled by syringing the right ear with colder water, a downward current is set up in the endolymph of the vertical canals. As the horizontal canal is lying in the horizontal plane in this position of the head, it will not be affected by these vertical convection currents. This downward flow towards the ampulla in a vertical canal, according to Ewald's first law, is a minimum stimulus for a vertical canal, and according to Ewald's second law a minimum stimulus in a canal produces nystagmus to the opposite side. According to Flourens' law, the vertical canals produce rotatory nystagmus. Therefore, following cold irrigation of the right ear when the head is erect there is a rotatory eye nystagmus to the opposite side.

This nystagmus, which is the result of sudden movement or temporary change of temperature, disappears when the effect of the movement passes or when the temperature becomes uniform. It may be thought of as a sudden readjustment of the animal's eyes to quickly counteract the movement of the head (or, in case of the caloric stimulation, to counteract the movement of the endolymph, which is interpreted as from a movement of the head), so that the original field of vision is maintained as far as possible. There is not a tonic effect upon the eye muscles as in the case of compensatory eye positions resulting from otolithic and neck reflexes. There is a rapid readjustment of the eyes, which is maintained by the otolithic and neck reflexes so long as the new position of the head is maintained.

Association of Semicircular Canals and Limb Movements.—McNally and Tait (1925) have attempted in the frog to analyze the exact relation between canal stimulation and the resulting effect on the body musculature. It has already been pointed out that Flourens and DeCyon found characteristic abnormal body reactions after injury to one or different combinations of the semicircular canals in the pigeon or rabbit. We

attempted to produce isolated lesions of the different ampullæ of the canals without injury to the adjacent structures. Special care was taken to avoid injury to the otolithic mechanism. These operated animals were observed over long periods to determine the exact effect the removal of one or any combination of ampullæ had upon the body, but more especially upon the limbs of the animal. (We did not study the eye reflexes, as they are difficult to observe in the frog.) We first made a careful study of the reactions of the normal frog to gravity—that is, to change of position, to centrifugal force, to turning about a vertical axis and to turning about the different horizontal axes. We found that when a normal frog is placed on a board and tilted out of the horizontal plane it readjusts its limbs and body so as to maintain the head as nearly horizontal as possible. For instance, if the animal is tilted forward the front limbs are extended and the head is raised. If the animal is slowly tilted backwards the front limbs are flexed and the head is gradually lowered until the snout touches the table.

When the normal frog is subjected to centrifugal force—that is, when placed on a turntable at a definite distance from the center and the speed of the table is so slowly increased as not to stimulate turning reactions, if placed side to the center, the animal gradually leans in towards the center in such a way as to protect itself from being thrown off, the limbs on the outer side of the body being extended, the limbs on the inner side being flexed.

When a normal frog is placed on a turntable and rapidly rotated it responds by walking around in the opposite direction, as though it were trying to retain its original field of vision. If stopped suddenly, it makes a circus movement in the direction of the turning. When a frog is placed on a tilt-table and rapidly turned about a horizontal axis, it executes rapid protective movements to avoid being thrown off. For instance, if tilted forward, there is a rapid extension of both arms and a raising of the head. If tilted backward both hind legs are thrown out rapidly, the arms are extended and the head is lowered. All these responses of the normal frog are exceedingly rapid and perfectly well timed. There never seems



to be an excessive movement in any direction, so that the normal animal does not overcompensate.

In our early experiments we attempted to cut the nerve to an ampulla, but as it is difficult to avoid pulling on adjacent structures we finally adopted cauterization of the ampulla as a safer method of functionally eliminating one semicircular canal. All operations were eventually confirmed by postmortem investigation of the labyrinth with a dissecting microscope and in a few cases by histologic serial sections of the labyrinth. For sake of brevity, it might be added that following a lesion of one or all six semicircular canals the response of the animal to slow tilting and to centrifugal force was perfectly normal. In other words, the gravity or positional responses were unaffected by the removal of the semicircular canals. When at rest the posture of all these animals was perfectly normal—that is, there were no forced positions, and, therefore, no change of muscle tone following a semicircular canal lesion.

**Destruction of the Left Horizontal Semicircular Canal.**—In jumping or swimming there is a slight tendency to turn to the left. On the turn-table this frog shows no response during turning to the left. When the turning is stopped suddenly, the animal makes circus movements to the left. During turning to the right, the animal walks around to the left. When the turning is suddenly stopped, the animal remains perfectly still. It would seem evident, therefore, the remaining right horizontal semicircular canal responds to turning in only one direction.

**Destruction of the Left Anterior Vertical Semicircular Canal.**—In jumping, this animal shows a tendency to turn to the left, and when landing it tends to land on the left shoulder as though the left arm were not in the proper position to receive the weight of that corner of the body. However, once landed, it promptly picks itself up and assumes a normal squatting position. When on the tilt-table and tilted rapidly in the plane of the injured canal, if tilted backward it reacts normally. If tilted forward the head goes down and to the left, the most evident thing being a lack of thrusting forward of the left arm. However, once the tilting is stopped and the animal is now out of the horizontal plane, the slower utricular

effect evidently comes in, so that the left arm is extended, the shoulder is raised and a normal posture assumed for the new position.

**Destruction of the Ampulla of the Left Posterior Vertical Canal.**—When jumping, the animal tends to turn to the left, and on landing it lands on the left posterior corner of the body, the left posterior limb not being properly placed to take the weight. When on the tilt-table and tilted in the plane of the injured canal, if tilted forward it reacts normally. If tilted backward, the body sags back, the left hind leg is not thrown out, the right leg is extended normally. It is an easy matter to topple such an animal over backwards. However, if the tilting is not too rapid, the left hind leg is extended when the tilting has ceased, the positional responses from the utricle coming in.

**Destruction of the Ampullæ of Both Anterior Vertical Canals.**—In jumping, the animal does not seem to raise the anterior part of its body properly, whereas the hind limbs kick off normally, with the result that the body is propelled along close to the ground. On landing, instead of the arms being prepared to receive the weight of the front part of the body, they are extended laterally so that the animal lands nose down on its belly. Once landed, it picks itself up and squats properly. In swimming, this animal dives to the bottom and swims along the bottom of the tank. On the tilt-table, when tilted back, the response is practically normal. If tilted rapidly forward, the body lurches forward, the arms are not properly extended. When it comes to rest, the arms are extended normally and the front part of the body is raised.

**Destruction of the Ampullæ of Both Posterior Vertical Canals.**—When jumping, this animal seems to put the normal amount of energy into the impulse from the front legs, so that the front part of the body is well raised. However, the hind legs do not seem to give their usually powerful kick forward at the proper moment, with the result that the body is thrown up into the air and often turns backward head over heels. In swimming, such an animal seems to be constantly trying to shoot up out of the water. On the tilt-table, when tilted forward, the response is practically normal. When tilted rapidly backward, the body slews backward, the hind legs are

not extended normally, so that the animal may be very easily tilted backwards. When the tilting ceases the animal assumes the normal pose for that new position.

Destruction of the Ampullæ of All Six Semicircular Canals. —Such an animal seems loath to jump. If forced to do so it sways markedly before taking off. When it takes off, it throws itself in the air and lands in any position. Once landed, it assumes a normal posture. In swimming, it sways from side to side as though not properly ballasted. On the tilt-table, if tilted rapidly in any direction, the body slews over in that direction and may be very easily thrown off. Once the tilting stops, the animal assumes a normal pose for the new position. From these experiments it becomes evident that following a lesion of one or of all six semicircular canals in the frog the response to position or gravity stimulation and to centrifugal force is normal. When the horizontal canals are intact, the responses to turning about a vertical axis are normal. When a vertical canal is injured, the animal loses its rapid protective responses to tilting in the place of the injured canal in the direction to which the ampullated end of the canal points. It seems likely, therefore, that each canal is responsible for the rapid protective reflexes in response to movement in its own plane. When the movement is in such a plane that two canals are involved they cooperate in bringing about the normal protective reflexes. When watching an animal with one vertical canal injured, the most evident thing is a failure to rapidly thrust out the limb of the corresponding corner of the body. However, there is also a failure or a lack of flexor tone in the contralateral limbs, which allows undue slewing of the body, etc. This would mean, therefore, that each canal governs the rapid movements of all four limbs, but in an asymmetrical manner, somewhat analogous to the way in which the neck reflexes influence the tone of all four limbs. As an illustration of what is meant, if we consider what happens when a normal frog is resisting tilting forwards, the normal response would be the extension of both fore limbs and flexion of both hind limbs. This is an asymmetrical influence coming from the anterior vertical canals. Again, when a normal frog is tilted laterally, say to the right, the right anterior and posterior vertical canals are being stimulated and the normal limb responses

consist of an extension of the right fore and hind limbs and a flexion of the left fore and hind limbs. This asymmetrical influence of the canals upon the limbs is well seen in a jumping horse. As the horse is raising off the ground, the posterior vertical canals are being stimulated, so that as he goes up into the air the front limbs are flexed and the hind limbs are extended. As he approaches the ground at the end of the jump his head is going forward, therefore, the anterior vertical canals are being stimulated, so that the front legs are extended and the hind legs are flexed.

McNally and Tait, in a recent set of experiments not yet completed, attempted to analyze exactly the limb and muscle response from stimulation of each semicircular canal, to see if it could be experimentally proved just exactly what groups of muscles are cooperating. The technic consists in destroying the ampulla in one vertical canal in the frog. The hind limbs of the animal are then operated upon to make the well known physiologic nerve muscle preparation—that is, the sciatic nerve is dissected from the other structures in the thigh, which are all completely removed just below the hip and above the knee. This leaves the lower segment of each hind limb connected to the body only through the intact sciatic nerve. We have designed a special small tilt-table, the axes of the table being attached to a stationary platform. Above this stationary platform are arranged four recording levers, which are in contact with a revolving drum. The body of the animal is fixed to the tilt-table, the sciatic nerves bridge the axis of the table and both lower segments of the hind limbs are attached by a pin through the knee to the stationary table. The flexor and extensor group of muscles of each lower limb having been previously dissected out, the tendons from each group are attached to one of the four levers. By this arrangement we have the flexor group and the extensor group of the lower segment of each hind limb recorded on the drum. It is possible to subject the body component of the frog to forward or backward tilting without disturbing the fixed hind leg segments. Any impulse which reaches these segments must come as a nerve impulse through the sciatic nerve. We have so far succeeded in proving that one vertical canal affects more than the limbs of the corresponding corner of the body, but as yet we have

not worked out all the details of the cooperation of the different muscle groups.

The semicircular canals initiate rapid readjustments of both the eyes and limbs in response to a sudden movement or change of movement. In the absence of all six semicircular canals the animal may still respond to excessively slow movements, such as slow tilting or gravity response, but as a protective reaction to a sudden stimulus this is of very little value because the animal would be thrown over before this slow reaction or reflex from the utricle would bring about the proper protective attitude. When the semicircular canals are intact and when the head is suddenly moved in any direction there is a rapid movement of the eyes in the opposite direction. By the time the movement of the head has stopped, the compensatory eye positions from the utricles and from neck reflexes have come into effect so that the eyes are maintained in the new position, which was rapidly assumed by virtue of the nystagmus which was initiated through the semicircular canal stimulation. When a normal animal is subjected to a sudden movement, as in jumping or stumbling, there is a rapid protective movement of the arms and legs. For instance, if one stubs a toe or trips, the arms are suddenly thrown forward; we say instinctively, because it requires no conscious effort on our part, but in reality the throwing forward of the arms is a rapid protective reflex from the anterior vertical semicircular canals, so that in case of a fall forward the arms are in a position to receive the weight of the upper or forward part of the body. This sudden movement of the arms is followed by the more slowly occurring utricular reflex, which maintains the limbs in the new position so long as the new position of the head is maintained. It is our semicircular canals, therefore, that allow us to make so many rapid corrective movements, which are ordinarily thought of as instinctive. In the case of the frog these same protective movements are made, even in the decerebrate animal. Eye nystagmus following semicircular canal stimulation is only a very small part of canal function. However, clinically, when thinking of diseases of the labyrinth, the tendency is to think in terms of eye nystagmus. It would seem that if a systematic examination was made for the presence of these other rapid protective movements of the limbs and

trunk that some abnormality might be detected, which would prove to be of even more diagnostic significance than the eye nystagmus. This would seem to be more probable in the case of an intracranial lesion which may involve only the fibers of one or other of the canals. In this way it might be possible, by studying these rapid protective reflexes, to arrive at a clinical diagnosis of a lesion of a single canal or of a pair of canals. This information might also help to determine the intracranial pathways from each ampulla.

#### SUMMARY.

Semicircular canals are organs of kinetic equilibrium—that is, they respond to sudden movement or change of movement.

There are three semicircular canals in each ear. The six canals are so arranged that in each ear there is a canal in the same plane but oppositely directed, the horizontal canals both being in the horizontal plane and one anterior vertical canal being in the same plane as the opposite posterior vertical canal.

The semicircular canals are stimulated by any sudden movement of the head, and they bring about a prompt readjustment of the eyes and limbs to counteract the movement of the head.

The semicircular canals have no tonic effect upon the body musculature.

#### LECTURE 5.

#### SOME CLINICAL APPLICATIONS OF THE PHYSIOLOGY OF THE INTERNAL EAR.

In this last lecture I will outline a simple and practical labyrinthine examination. With this as a basis, the examination can be made just as complete as the examiner wishes. This is just the outline. I will then refer, if you will bear with me, to some of the clinical remarks already made, and I would like just to mention some of the questions concerning both the cochlea and the vestibule, about which there is no general agreement.

**A Simple and Practical Labyrinthine Examination.**—After taking a careful history, particularly for details of deafness, noises, dizziness, etc., one should proceed to a routine examination of the ears, nose and throat, special attention being given to the condition of the ear drums and middle ears and

to movements and sensations of the larynx, soft palate and facial muscles. The presence of spontaneous nystagmus, past pointing, incoordination and Rhombergism, with the head in different positions to detect utricular diseases should be investigated. Examination of the hearing, especially for bone conduction, should be done, and, if possible, confirmed by an audiogram. This may be followed by the cold caloric test, using ice cold water (if the drum is not intact, use Dundas Grant's cold air douche). Immediately after an ear is irrigated the eyes are examined, both with head erect for the vertical canal response, and with head back for horizontal canal response, and in each case past pointing should be examined and dizziness inquired about. The rotation test may be done for the horizontal canals, especially if any abnormality has been noted following caloric stimulation.

When carrying out a caloric or rotation test where there is a spontaneous nystagmus present the patient should be made to look in that direction in which the spontaneous nystagmus is absent or at a minimum; then any nystagmus which follows or is increased after stimulation is a result of the stimulus and a direct indication of the labyrinthine condition.

This examination is very brief and will not require more than one-half to three-quarters of an hour (not including an audiogram), and for routine use it must be brief, else the practicing specialist has no time to devote to it. I have tried to show that an examination of the internal ear may not be too detailed and yet be of some value in diagnosis.

In assessing the value of either the vestibular or cochlear tests they must be considered in conjunction with each other. Preferably there should be an audiogram to show the degree of deafness and the part of the scale most affected. If there is no evidence in the middle or internal ear of acute or chronic suppuration or of other local disease, such as otosclerosis, which may be accompanied by vestibular symptoms, and there are signs of labyrinthine irritation or nerve deafness, such as dizziness, deafness, nausea, etc., a very careful general physical examination should be carried out for diseases situated elsewhere in the body, which may be classified under the following headings:

1. Diseases of the central nervous system.
  - (a) Brain tumor and brain abscess.
  - (b) Nervous diseases—Disseminated sclerosis, etc.
2. Trauma—Fracture of the skull involving the petrous bone.
3. Vascular disease—Arteriosclerosis, anemias.
4. Deficiency diseases—Osteomalacia, etc.
5. Specific infection—Tuberculosis, syphilis.
6. Toxic conditions—
  - (a) Endogenous—Foci of infection and intoxication.
  - (b) Exogenous—Lead poisoning, arsenic, alcohol, tobacco, etc.
7. Congenital defects of internal ear.

If anything is found during the general physical examination, which should include the routine blood chemistry, blood Wassermann and basal metabolic tests, this should receive immediate treatment in the hope that it may have a curative or at least an arresting effect on the local ear condition, because so very little can be done at present in the way of treatment to the ear itself.

Having outlined a fairly simple but, for practical purposes, a moderately thorough examination of the internal ear, I would like to discuss some of the individual tests and some ear diseases about which there is still a wide difference of opinion among otologists. First, some questions relative to the vestibule and finally some of the cochlear difficulties.

Clinical Application of the Vestibular Mechanism.—The clinical investigation of the labyrinth is more or less confined to the observation of the eye reflexes following labyrinthine stimulation, as they are the only ones that can be studied with any degree of ease and certainty. Up to the present, tests for detecting otolithic diseases are very uncertain. Care must be taken to exclude neck reflexes and cooperating reflexes from other sense organs. Examination of the utricle should be made with the patient lying in different positions, the neck and body being held rigid and symmetrical to eliminate neck reflexes, etc., in order to bring out the pure labyrinthine—utricular—effect. According to Magnus, the maximum utricular effect, which is increased extensor tone of all four limbs, should be obtained from the patient lying on his back, the head



being bent back so that the line of the mouth forms an angle of  $45^\circ$  above the horizontal plane. The minimum utricular position is obtained when the patient is lying prone with the head bent forward so that the line of the mouth forms an angle of  $45^\circ$  below the horizontal plane, the effect being increase in the flexor tone of all four limbs. Then careful examination should be made to detect any compensatory eye movements, nystagmus, dizziness or change of muscle tone in the limbs, etc. Any change noted must be purely dependent on the position assumed and should last only so long as this position is maintained. Care must be taken to exclude disturbances which are perceived by the patient or by the operator during the actual assuming of any position. Therefore, when a position is assumed, a few minutes should be allowed before any observations are made. If under these conditions any of the above signs or symptoms are noted, they would indicate a disturbance of utricular function. One of the commonest symptoms reported when the patient is placed in a certain position is that nystagmus occurs when the position is assumed. If care is taken to eliminate the possibility that the act of assuming the position is not responsible for the nystagmus, the nystagmus is usually ascribed to the otolithic mechanism. However, from the experimental evidence mentioned, it will be remembered that a pure lesion of the saccule or utricle does not cause nystagmus in the experimental animals. This would suggest that some other part of the labyrinth, not the otolithic mechanism, will be found to be the seat of origin of this nystagmus, which occurs when a certain position of the head is assumed. As yet there has been no general acceptance of any practical test to detect a loss of response to gravity stimulation which would indicate loss of utricular function, such as is elicited by slowly tilting the delabyrinthized frog out of the horizontal plane. Many instruments have been devised to detect compensatory eye movements in the human, but so far the instruments are all complicated and expensive, and, therefore, not practical. Moreover, the test was understood to give evidence regarding the state of the saccule. Experimental evidence, however, suggests that the saccule has no control over eye movements or positions. All compensatory eye positions disappear after destruction of the utricular nerve. These tests

will certainly have to be revised and their diagnostic significance, as is generally understood, changed before they can be retained as otologic tests. Before general acceptance can be hoped for, they will also require simplification.

Quix has outlined an elaborate test for detecting saccular diseases, but, as was pointed out, there is not yet definite experimental proof that the saccule possesses any equilibrical function. Until the physiology of the saccule is definitely established, clinical tests are of little value. Certain changes in the compensatory eye positions have also been attributed to the saccule, but those also must be considered as doubtful in view of the negative experimental findings.

The semicircular canals have long been used clinically as a diagnostic aid to the condition of the labyrinth. Bárány improved this method of examination. Up to the present the method consists of stimulating individually the canals as far as possible, and then observing the effect upon the eyes—nystagmus. As yet, apart from past pointing, no widespread attempt has been made to arrive at a knowledge of the condition of the semicircular canals by an investigation of the behavior of the limbs, etc., during turning or tilting out of the horizontal plane. The common examinations for clinical function are the electrical, rotation and caloric tests. The electrical test is not generally used as a routine procedure, as it is more a test of the condition of the eighth nerve proper. The rotation test, which is carried out in a specially designed chair, has the disadvantage of stimulating both labyrinths at the same time. As generally used for the vertical canals the rotation test gives very little definite information because due regard is not taken of the actual planes in which the vertical canals lie. Putting the head forward or backward does not stimulate either the anterior or posterior canals in any characteristic manner. To attempt to stimulate the vertical canals by rotation, a pair must be chosen which lie in the same plane, and the head must be so arranged that this pair of canals are brought into the horizontal plane—that is, at right angles to the axis of rotation. As pointed out by Crum Brown, an anterior vertical canal of one side is in the same plane as the posterior vertical canal of the opposite side. Therefore, to stimulate the right anterior vertical and the left posterior vertical canal, which are in the

same plane, and to bring these into the horizontal plane, the head must be tilted forward  $90^\circ$  and then rotated  $45^\circ$  to the left so that the occiput is turned to the left shoulder. For the opposite set of vertical canals, after tilting the head  $90^\circ$  forward the head must be rotated to the right  $45^\circ$  so that the occiput is turned to the right shoulder. As these positions are uncomfortable for the patient and difficult to gauge, the rotation test is of most value when used for the horizontal canals.

The caloric test is really the most practical of all. Since Kobrak has introduced the fractional method of procedure this test has gained much in clinical value. The application of this test varies with the individual. The procedure in Dr. Birkett's clinic is to use ice cold water. This eliminates the necessity of taking the temperature and allows for the use of a small quantity of water. It is found that in the majority of cases, using a small syringe and a needle to direct the flow, that 2 cc. of ice cold water carefully directed against the drum gives a definite nystagmus in 15 to 30 seconds with very slight or no dizziness, and the nystagmus lasts from 60 to 120 seconds. This method is practical except where there is a quiescent chronic suppurative in the middle ear with perforation, etc. In these cases Dundas Grant's cold air douche may be used. For practical purposes it is found that blowing the cold air against the drum for about a minute is sufficient in the normal ear to bring about a response.

Interpretation.—A simple method of interpreting the results of semicircular canal stimulation is by making use of the laws of Flourens and Ewald. The results that should follow any semicircular canal stimulation can be thus determined. For example, when the head is erect, if the right ear is syringed with cold water, there is cooling of the endolymph in the labyrinth and convection currents are started in a downward direction. There is, therefore, a downward flow of endolymph in the right anterior vertical canal and probably in the right posterior vertical canal—that is, a flow towards the ampulla. As the flow is in the vertical canals, the nystagmus will be rotatory (Flourens' law). Flow towards the ampulla in a vertical canal is a minimal stimulation (Ewald's first law); nystagmus, therefore, will be to the opposite side (Ewald's second law); thus the effect of syringing the right ear, head

erect, with cold water should be rotatory eye nystagmus to the left.

A simple rule for interpreting past pointing, falling and vertigo is to remember that normally falling and past pointing are always in the direction of the endolymphatic flow, whereas vertigo is always in the opposite direction to the endolymphatic flow, or, otherwise stated, the vertigo is in the direction of the nystagmus, and past pointing and falling are in the opposite direction to the nystagmus.

By using a small quantity of water in the caloric test a more accurate knowledge of the state of irritability of the labyrinth can be arrived at. In some cases a violent nystagmus and dizziness is produced by syringing the ear with one-fourth of a cc. of ice cold water—hyperexcitability. In other cases it may require 30 cc. of ice cold water to elicit a response—hypoirritability. Hyperexcitability probably occurs in the very early stages of labyrinthine irritation and disease.

According to several authors, Lewis, Lewitt and others, hyperexcitability of the labyrinth, as evidenced by an exaggerated response—increased nystagmus, vertigo, etc.—is suggestive of a supratentorial or cerebral lesion. If the lesion is old standing, however, the hyperexcitability gives way to hypoirritability. The underlying idea is that when the nystagmus and vertigo, etc., are normal in character, even though increased in amount, the pathways in the brain stem must be intact to allow of the connection with the external ocular nuclei. The increased irritability of the labyrinth in cerebral diseases probably means pressure on or beginning involvement of the intracranial pathways from the labyrinth. Together with this symptom of cerebral disease may be considered the test recently suggested by deKleijn.

In the case of a tumor of the cerebrum, frontal or temporal lobe, the quick phase of nystagmus is increased to the side of the lesion. Therefore, when the ear on the side of the lesion is irrigated with warm water or the ear on the opposite side is irrigated with cold water, each will be followed by an exaggerated response—nystagmus to the side of the lesion. Whereas, when the ear on the side of the lesion is irrigated with cold water or the opposite ear is irrigated with warm water, each will be followed by a diminished response—nys-

tagnus to the opposite side. deKleijn has found this test reliable in several proven cases. It was my privilege to assist him with the testing of some of these cases.

A syndrome which seems to receive support from many authors, Lewis, Wishart, Lewitt and others, is that in the case of a cerebellopontine angle tumor, there is an almost totally destroyed ear on the side of the lesion, with loss of response or diminished response from the vertical semicircular canals of the opposite side, whereas the horizontal canals of the opposite side respond normally. This means no response from the vertical canals of either side, and no response from the horizontal canals on the side of the lesion. If it is an eighth nerve tumor this would suggest the tumor was involving the brain stem either directly or by pressure.

The rotated position of the head, commonly called the cerebellar position of the head, probably signifies a lesion of a utricular macula or of its intracranial pathways. When present in cerebellar disease it probably signifies pressure on the eighth nerve if the rotation is to the side of the lesion, and it probably signifies pressure on the pons and midbrain, if the rotation of the head is to the side opposite to the lesion. A lesion of the internal ear or of the eighth nerve produces rotation of the head to the same side. According to Muskens, a lesion of the pons or midbrain leads to a rotation of the head to the side opposite to the lesion. It will be seen, therefore, that apart from suggesting a lesion of a utricular macula or its intracranial pathways, this sign has very little localizing value in intracranial diagnosis. It must be remembered that peculiar head positions also follow ocular paralysis.

It might again be in order to briefly refer to the diagnostic significance of vertical nystagmus. In order to have a vertical eye nystagmus we must have a combined effect from either both posterior vertical canals or else both anterior canals acting at the same time. It is rare to have a clinical lesion so symmetrically situated in both ears. It is easier to understand a lesion involving the intracranial paths from both anterior vertical or both posterior vertical canals at the same time, where they are close together in the brain stem near the midline. For this reason, a vertical nystagmus is considered diagnostic of a brain stem lesion.

Clinical Application of the Cochlea.—The chief value attached to the hearing tests is to decide whether a lesion affects the cochlea or perception mechanism or whether it affects the middle ear or sound conducting mechanism.

The main point in differential diagnosis is usually based on the fact that relative bone conduction is increased when there is a lesion of the conductive apparatus, whereas, both air and bone conduction are reduced when there is a lesion of the perceptive apparatus. The chief loss for air conduction of a vibrating tuning fork in a lesion of the perceptive mechanism is for the high tones. The audiogram shows a falling off in the high notes. Yet why this should be so does not seem clear from the physiology of the cochlea, unless it is because the area of the cochlea where high notes are received is at the base of the cochlea, just behind the promontory, and therefore most affected by local disease. However, this seems an unsatisfactory explanation.

Testing for absolute bone conduction—i. e., where both meati are closed, which equalizes all errors in the conductive system, would seem to hold promise of being a good test for perception deafness. Because by this method all vibrations from the fork on the bone are thrown back into each internal ear, and, therefore, any failure to perceive the sound in one ear would suggest a lesion of the perceiving mechanism. In this method of testing, the conductive lesion is decided from a consideration of the interference with air conduction of the lower sound vibrations. An ideal arrangement would seem to be the testing for both relative and absolute bone conduction—one giving some information regarding the perceptive mechanism and the other regarding the conductive mechanism for sound.

As at present carried out, it is very difficult in many cases to decide, after a careful hearing test, just whether the lesion is in the conductive or perceptive mechanism.

There are many interesting conditions, which may be briefly considered.

In carrying out an examination of hearing, especially in a patient over 50 years of age, it is always difficult to decide how much loss should be allowed for senile atrophy. Just how much loss of hearing should be allowed depending upon the age of the patient has never been definitely determined. With

the general use of the audiometer, however, it will be possible to determine the normal decrement of hearing for a given age. In Johns Hopkins, Bunch has studied audiograms made in a large series of normal cases, over 350. They have divided these cases in decades according to their age. The mean audiometer curve for each decade is thus arrived at. This data will serve as a fairly accurate basis upon which to compute the allowance of hearing loss that should be made for a patient in any particular decade of life. The curve remains pretty close to the normal adult curve until the decade between 40 and 50 years of age is arrived at. The curve then begins to drop, there being a definite falling off for the high notes 4096 and 8192. As the age increases, the falling off in these high tones is more marked. However, up to 60 years of age 8192 double vibrations are still heard by the majority of cases. For the low notes up to 1024, the curve remains pretty much the same for all ages—i. e., between 15 to 25 sensation units. It would seem, therefore, that any marked falling off for these low notes in an older individual would suggest a pathologic cause other than mere senile change, or a complete loss of the higher notes would suggest a cause other than senility.

Otosclerosis.—Otosclerosis itself is becoming increasingly difficult to diagnose. The classical symptoms, the triad of Bezold—loss of low tones, increased bone conduction and negative Rinne—is only present in a very small percentage of the cases. These symptoms are due to fixation of the footplate of the stapes in the oval window. This fixation occurs when the otosclerotic patch develops at what is called the site of election—that is, at the anterior margin of the oval window, bringing about a bony ankylosis of the stapes. If the otosclerotic patch is present in this location and yet has not involved the annular ligament of the stapes, the case would not show the triad of Bezold. It is simply an exaggerated type of lesion of the sound conducting mechanism. This same triad is present if the stapes is ankylosed by fibrous adhesions following acute or chronic suppuration in the middle ear. If the otosclerosis begins in any other part of the bony capsule of the internal ear, the symptoms depend upon the structures affected at the site of the lesion. If it is in the vestibular portion of the labyrinth, the first symptoms might be nystagmus,

dizziness, etc. Again, if the otosclerotic patch first involves the modiolus, the deafness is of the character of a lesion of the sound receiving apparatus or nerve deafness, so that Bezold's triad is only present when there is fixation of the stapes.

Gray claims that as the onset of otosclerosis is associated by a local vascular change, there is also an alteration in the vascular supply to the spiral ganglia, which causes a certain amount of atrophy or degeneration of these cells. Gray explains momentary improvement in hearing, which does occur in the course of otosclerosis, as due to a sudden temporary improved blood supply to the cells of the spiral ganglia, which allows of better conduction of the nerve impulses at that moment.

It would seem evident, therefore, that in the majority of cases a positive diagnosis of otosclerosis may be impossible for a long time, for even a period of years, because the progress of the condition is very slow. This would suggest that it is almost impossible to be sure, from the clinical examination, that one is dealing with an early case of otosclerosis, so that even in the so-called early cases cure may mean overcoming advanced bone disease.

**Catarrhal Deafness.**—When the loss of hearing is definitely of the character of middle ear deafness and not definitely otosclerotic, in the absence of acute or chronic suppuration, a catarrhal condition of the eustachian tube or middle ear cavity is usually presupposed. It is usually called catarrhal deafness. If the deafness is caused by a slight obstruction to the eustachian tube, inflation of the tube by Politzer's method tends to equalize the pressure on both sides of the drum membrane and the hearing may be improved. If this congestion is due to some abnormal condition of the nose and throat, such as adenoids, deviated septum, etc., this activating condition should be removed. This type of deafness is found most commonly in children, where the results from treatment are most gratifying. However, when there is a deafness of middle ear character present in adults, and there is no history of evidence of suppuration, slight abnormality in the nose or throat, and not sufficient evidence for a definite diagnosis of otosclerosis, the condition in the majority of cases is considered to be one of chronic catarrhal otitis media. As the name suggests, this means chronic or recurring catarrhal inflammation in the mid-



dle ear in the absence of definite suppuration. In the more severe cases, the pathology is supposed to be fibrous adhesions in the middle ear between the ossicles, drum membrane, etc., and fibrous strictures in the eustachian tube. These cases are usually treated by Politzerization, catheterization of the eustachian tubes, and dilatation of the supposed strictures with bougie, etc. Very seldom is there any improvement in the hearing.

It is very probable that we are blaming the eustachian tube and middle ear for the deafness, when in reality we are unable to discover the actual causative lesion. In this class of case, if the true diagnosis could be arrived at, some progress might be made in treatment. At least, when a definite diagnosis of otosclerosis cannot be arrived at, one feels that something more amenable to treatment may be the cause.

Progressive Deafness.—There is a type of deafness which occurs in young people between the ages of 20 and 25 years, who are apparently in good general health. The symptoms are not typical of either middle ear or nerve deafness. Many are the vigorous athletic type. In this class there is no family history of deafness. When they report for treatment the usual history is that for the past year or two they have noticed a slight deafness in one or both ears, and there may or may not be tinnitus. On examination, the audiogram shows about a 25 to 40 per cent loss of hearing, usually throughout the whole scale. In some cases, however, the chief loss may be in the low tones; in other cases, in the high tones. The bone conduction remains about normal. The eustachian tubes have normal patency. There is no definite history of recurring nose and throat diseases, and no evidence of nose and throat pathology. In the majority of instances, the labyrinth or vestibular portion of the internal ear is normal. When subjected to a complete general physical examination, including basal metabolism and blood chemistry, nothing definite can be found. Blood Wassermann is negative; blood calcium is normal. These patients are not improved by Politzerization. The deafness seems to be of the progressive type. These patients are not typical of nerve deafness or of middle ear deafness. Will they eventually show some evidence of otosclerosis, or are they early cases of nerve deafness from an unrevealed toxic focus? If

the true etiology of the deafness could be detected at this relatively early stage it is just possible that something might be done to prevent or retard the progression in these cases also.

It may be suggested that these are cases of chronic catarrhal otitis media or of chronic adhesive deafness, but in the cases referred to there is no history of recurring colds, etc. The drums look practically normal—there is no undue retraction. The eustachian tubes are open; the nose and throat are practically normal.

These cases are usually considered under the heading of catarrhal or progressive deafness. This last seems a less confusing name for this indefinite type of case, because it does not suggest a diagnosis.

It is becoming increasingly evident that the most satisfactory method of treating deafness is to detect it in its earliest beginning in early childhood. To this end group testing of hearing in children in the schools for selection of the early deaf cases will be of very great benefit. The deaf children are selected and are referred to otologic clinics for proper treatment in the hope that it is not too late for a cure, or at least not too late to prevent progression.

Syphilis of the Internal Ear.—It was for a long time considered that syphilis of the internal ear was a definite entity. We were taught that from a clinical examination of the ear we should be able to make a diagnosis of syphilitic nerve deafness. In a recent paper, Alexander illustrated the uncertainty of our knowledge of this condition. He reports six cases of syphilis in which there was well marked deafness. Histologic section of the ear was carried out in all cases. In each of the six cases the pathology found was of a type usually associated with some other condition, otosclerosis, Paget's disease, increased intracranial pressure, etc. In no case was there a typical lesion of syphilis.

The facts brought out in this series of cases would seem to suggest that our present knowledge does not allow of a definite diagnosis being made of syphilis of the internal ear.

It was formerly taught that a very severe loss of bone conduction in a young individual was very characteristic of syphilitic deafness. In two cases of deafness now under my care—both married women, one aged 37 and the other 38 years—a

complete examination of the hearing was made. The onset was about two years previously; no family history; no nose or throat trouble; eustachian tubes open—hearing not improved after inflation. Drums practically normal. The audiograms showed an average 35 per cent loss of hearing in one of the cases, with complete loss for 8192, and in the other an average loss of 30 per cent, the curve dipping down at the high notes, 8192 just being heard. In both cases relative bone conduction by Schwabach's test is normal for each ear. Both these cases would fall into that indefinite class mentioned before. One of these patients was given an exhaustive general physical examination, including blood chemistry, basal metabolism, blood calcium and blood Wassermann. Everything was normal, except the blood Wassermann, which was four plus. The other patient has not been examined generally, but she admits having been treated for syphilis. We have, therefore, two cases of deafness with normal bone conduction, an air conduction loss of about 30 per cent, with a tendency to go off on the high notes, and a luetic history in each case. If this is syphilitic deafness there is no loss of relative bone conduction. It would be impossible to diagnose syphilitic deafness from this otologic picture.

#### SUMMARY.

1. The cochlea is the end organ for the perception of sound vibrations transmitted to it by the intact drum and ossicles and through the cranial bones.

2. The human cochlea acts as a stringed resonator, the fibers of the basilar membrane being varied for length, tension and load, so that they are capable of responding to a range of sound of about eleven octaves, which is the normal range of human hearing.

3. The sound is analyzed into its simple harmonic constituents in the cochlea, and the nerve impulses from the corresponding sectors of the basilar membrane are transmitted to the brain.

4. There are three semicircular canals and a utricular and a saccular macula in each ear. The six canals are so arranged that in each ear there is a canal in the same plane, but oppositely directed—the horizontal canals both being in the hori-

zonal plane, and one anterior vertical canal being in the same plane as the opposite posterior vertical canal. The utricular macula lies in the horizontal plane, the saccular macula lying in an almost vertical plane.

5. The semicircular canals respond to sudden movement or change of movement—acceleration—and they elicit sudden readjustment of the eyes and limbs to counteract the movement of the head. They have no tonic effect upon the body musculature.

6. The utricular macula responds to change of position—gravity and centrifugal force—and it elicits slowly and maintains a readjustment of the eyes and limbs to counteract any movement of the head, so long as the new position of the head is held. It exerts a tonic influence on the body musculature—the limbs, the eyes and the muscles of the opposite side of the neck.

7. The saccular macula has not yet been demonstrated by experimental evidence to have any vestibular function.

8. The saccus endolymphaticus is probably an absorption chamber for the endolymphatic fluid.

9. When the head is suddenly moved, the semicircular canals elicit a sudden counter movement of the eyes and limbs, the new attitude assumed being indefinitely maintained by the utricular macula and cooperative reflexes from other sense organs of the body so long as the new position of the head is unchanged.

## Editorial

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During the last twenty-five years something worth while has been done to improve undergraduate schools of medicine. This has been brought about by moral suasion and, that failing, sterner methods were resorted to.

During the coming years we hope that much will be done to improve graduate teaching and schools, and much can be done by the same methods that have proved successful in undergraduate institutions. While it is true that any graduate in medicine may without further study practice in any field as a specialist, very few if any pursue that plan in the department of otolaryngology. In discussions on the question, the statement is almost invariably made that men cannot be compelled to qualify by resorting to any particular instruction or what might be decided upon as a minimum requirement. But would it be necessary to resort to compulsion? Would not a sufficient number be glad to attend were the necessary schools and data regarding them available?

We know that many practicing otolaryngology have not had and cannot get the grade of training that they want and that the country is capable of giving. We know that much inferior teaching is being done and that few of those who are teaching know what others are doing. The better grade of schools and the incompetent are unknown to each other as such. None know these facts as well as otolaryngologists, particularly otolaryngologists who take a healthy and hearty interest in the profession, and the public needing services. Ethically, in fairness to their public, the profession may rightly be expected to furnish it with protection in so far as reasonably lies within its power.

Those who are interested in seeing a better state of affairs ensue must recognize that a great diversity of circumstances compel men to use devious ways for satisfying their needs. Different environments, differing social problems of different men require that all cannot bend to the same qualifying routine.

As said by Dr. William D. Cutter, some years ago, the young man before beginning practice with time and no money

can easily trade his time in return for two or three years' instruction, but the man who has been in practice for a few years who has some money and little time will get the most he can in the shortest possible time. It is our responsibility to direct him where his needs can be met consonant with reasonable preparation and instruction.

For years to come both types will seek instruction, some, especially of the latter, to utilize their knowledge in the general field of medicine, while others will make otolaryngology their life work.

We should make it unnecessary for conscientious men to resort for instruction to incompetent schools. If we cannot raise the moral standard of men we can, in a relatively short time, raise the teaching standard of a sufficient number of schools to supply the demands of students.

Large institutions with teaching facilities that are content to train but five or six interns yearly are not doing their duty and are following the easiest line. They are not helping to solve the bigger problem. Were such training available for all the men necessary to meet the public need, the matter of providing teaching institutions would be relatively simple. In our present stage of development some large institutions carrying varying numbers on their staffs as interns are giving the best training, and that training is for that particular and fortunate group pretty generally satisfactory. Such potential otolaryngologists and the public whom they serve do not need help, or, at least, others are in more urgent need.

A third group—practicing laryngologists desiring to see advanced work visit foreign countries. No systematized instruction for such men is obtainable in this country, or if such is available it is not generally known. A central bureau of information could be established where all available data applicable to all three groups could be filed and from which authentic information would be sent to inquirers.

The Board of Otolaryngology, by requiring that men be tested by examination, is contributing, at what must be a great personal loss and sacrifice, to a faithful and useful service that is resulting in many men attempting to reach this standard.

In addition to this, however, we need to provide means by which that standard may be reached or surpassed.

The point is that we have standards as manifested by examinations, but we have not provided the means by which a sufficient number of men may reach those standards. No men need take examinations. Many will not, but many such would accept good training did they know where to get it, and, having obtained it, they would be more willing and able to successfully meet the test of examinations. Not only is it beneficial that men who are qualified to meet a certain standard should be known, but our institutions should be listed and graded and compelled by the force of professional opinion to supply facilities that would meet at least a minimum requirement. It should be a matter of record whether individuals and institutions advertising certain schedules are seriously putting such schedules in practical effect. It should be known what every individual and institution are teaching, whether they are advertising schedules or not. Men honestly endeavoring to equip themselves to the best of their ability believe that the information they have directs them to the best equipped teachers or teaching bodies, and they have no means of finding out where or if better are in existence.

Much can be done by informing graduate schools regarding the work of the others and in directing prospective students through a central bureau, perhaps, to institutions known to be successfully achieving reasonable results in the training of men for our specialty.

One of the most difficult problems met with by those who are giving time and thought and teaching hours to this question is how best to furnish men with a practical training in major operating.

With some such schedule as the following:

Nose and throat instrumentation.....	4 hours
Nose and throat physiology.....	10 "
Nose and throat dry and wet anatomy.....	24 "
Lectures—Embryology, histology .....	48 "
Pathology and bacteriology.....	48 "
Nose and throat clinic, diagnosis and treatment, A. M. and P. M.....	200 "
Dissection—Head, neck, thorax and brain.....	40 "
Neurology as applied to nose, throat and ear.....	16 "
Therapeutics .....	8 "
Ward rounds, quiz .....	32 "

Nose and throat operations in 16 weeks, 12 hours; tonsils, adenoids, turbinotomies, submucous resec- tions, etc. ....				352 hours	812 hours
Ear anatomy .....				24 hours	
Ear clinic .....				96 "	
Acoustics, quiz .....				16 "	
Allergy .....				8 "	
Functional—Examination and treatment of patients				56 "	
Ear operations on cadavers.....				24 "	
Diagnosis and treatment of labyrinthine intracra- nial diseases and new otology.....				20 "	244 "
Bronchoscopy, 3 hours weekly for 8 weeks.....				24 hours	
X-ray diagnosis .....				4 "	
Plastic nasal surgery.....				16 "	
Conferences .....				8 "	
Nose and throat cadaver .....				36 "	90 "
Library .....					
					1146 hours

Covered in eight months or one academic year,

a very fair foundation is laid, but it lacks what all schools of the kind lack—i. e., provisions for supplying facilities for teaching major operating.

This fault could be corrected in part and perhaps altogether by making a survey of all hospitals requiring otolaryngologic interns which in turn could obtain their supply from among the group having taken some such basic training as the above. Certainly from such material better qualified men could be obtained than by utilizing the present methods of choosing interns, and the finished product would be better balanced and superior to the one now turned out. Better laboratory, cadaver, diagnostic and dissection instruction would be had, and, were it known that institutions favored men who had taken such basic training, the best men would attend such preliminary training centers. Many smaller hospitals could be found able to utilize such material to advantage, and although many would be unable to get placed in the larger institutions they could in fairly large numbers be placed with older men in the small hospitals. Even if they did not get an opportunity to do major operating themselves, acting as first assistant to some older, better qualified mentor would be an advance and a great safeguard to the



public. At present, men go out from our many schools, speaking of type 2, without having had close up instruction on major operating on the living. When they begin to practice, if it is possible and they are conscientious, they will get an older man to guide them in their first major operations, but in many cases this is not in their opinion good policy or even impossible, and they go it alone with what must be frequently unsatisfactory and maybe dire results.

By organization and zoning the country it would seem that all schools could be open to the inspection of local qualified and appointed representatives. No school with teachers in good standing could refuse to submit to inspection.

Another advantage accruing from a general knowledge of what the other fellow is doing would result from utilizing the information acquired for the interchange of students. One school excelling in advanced laboratory qualifications having university research facilities would take students from one strong in diagnostic material, anatomic department, or what not.

The above plan in no way interferes with the special courses given in the many subjects that go to make up otolaryngology, nor does it interfere with the long courses designed to particularly qualify men seeking special degrees, etc., but it is a suggested plan aimed to supply a present public and professional necessity.

#### SUMMARY.

Many practicing laryngologists have not had and cannot get the grade of training that they want and need.

Institutions do not know what other institutions are doing.

The public may rightly expect the profession to protect it.

Means should be provided by which men be given the opportunity to attain a certain standard.

Graduate schools should be listed and graded.

An index should be made of all hospitals requiring otolaryngologic interns, where men qualified in basic subjects be supervised while doing or assisting at major operations.

All schools should be open to inspection of local qualified representatives of otolaryngology.

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## Abstracts of Current Articles.

### **The Diagnosis of Frontal Lobe Tumors.**

*James Collier, Brit. Med. J., 2:289-291, August 17, 1929.*

The signs for the localization of frontal lobe tumors may be divided into five classes:

1. The negative signs.
2. The contiguous signs.
3. The signs which have been deemed peculiar to, or especially marked in prefrontal lesions.
4. The signs peculiar to lesions within the anterior fossa of the skull.
5. The signs revealed by ventriculography. Those of the first class are important because tumors of the cerebellar, occipital, parietal, central and left temporal regions give no unmistakable signs of their position.

The contiguous signs are those produced by the extension of some process connected with the growth, pressure, edema or vascular disturbance into the neighboring convolutions. Thus, we may have some motor weakness—interference with speech. The difficulty to recall words means a lesion far back in the temporal lobe.

Among symptoms peculiar to lesions of the frontal lobe are:

(a) Mental symptoms, loss of reserve, childishness, change in character, loss of memory, etc.

(b) Frontal tremors which may be homolateral or contralateral and usually mean a temporal lobe tumor.

(c) The grasping and groping reflex (inability to let go of an object unless they forget that it is in their hand).

(d) Conjugate deviation of the head and eyes, when persisting in the absence of other localizing signs, may indicate a frontal or temporal lesion. When present it will help lateralize the tumor as a persistent deviation to the contralateral side or of weakness in deviation to the ipsilateral or homolateral side.

(e) Loss of diminution of the trunk reflexes is of lateralizing value and indicates sometimes a frontal location occurring on the contralateral side. It usually means a temporal location

and is probably a contiguous sign resulting from pressure on the pyramidal mechanism.

(f) Incontinence, present in 20 per cent of temporal tumors.

(g) Papilloedema, usually greater the further back the tumor. This is of no localizing or lateralizing value. Many frontal lobe tumors show no papilloedema.

(h) Lethargy of a deep but rousable kind occurs with many frontal tumors.

(i) Fits and status epilepticus may occur.

Signs peculiar to the anterior fossa of the skull are those of pressure upon the olfactory tract, the optic nerve or chiasma, or upon the nerves and vessels passing through the sphenoid fissure, and those of bony involvement. These are always certain localizing and lateralizing signs, but they only obtain with a hard tumor coming to the surface of the orbital lobe, or with a growth of the meninges or bone.

There then remain the signs revealed by ventriculography.  
M.

#### **The Relation of Ear Conditions and Bathing.**

*W. I. Daggett and R. Cove-Smith, Brit. Med. J., 2:296-297, August 17, 1929.*

From the point of view of the ear, nose and throat surgeon, the bathing community can be divided into four classes:

1. Healthy persons.
2. Those with septic nasal conditions (colds, etc.).
3. Those with a perforation of the membrana tympani.
4. Those with recurrent dermatitis or furunculosis of the external auditory meatus.

In normal individuals, deafness or tinnitus may result from the introduction of water into the meatus. This is generally due to cerumen, which may expand and press on the drum or may dam water back and thus occlude the meatus. Vertigo is possible where one meatus is occluded. Acute otitis media from the entrance of infected water into the eustachian tube occurs all too frequently. In public baths all precautions to prevent the water from being contaminated should be employed, such as the use of disinfectants, continuous filtration, frequent changes of water, careful scouring of bath, compelling bathers to scour with soap under a shower before entering

bath; preventing people walking along the sides of the bath with street shoes on, etc.

As a rule, it is the badly taught, inexperienced swimmer who develops an acute disturbance of the ear. The practiced swimmer breathes through his mouth at regular intervals and thus shuts off his nasopharynx and eustachian tubes. But the inexperienced swimmer gulps, chokes and swallows, thus opening up the tubes to the infected water.

Therefore, the public should be instructed to breathe properly in the water. Ducking people or pushing them in unawares is also unwise. Jumping into water from a height without holding the nose may cause water to be forced into the sinuses and tubes.

The second class of people—those who suffer from a septic nasal condition—not only spread infection but may also infect their own accessory sinuses or eustachian tubes.

Patients who have a perforated membrana tympana should not bathe. Those in whom the discharge has ceased will almost certainly become reinfected, with consequent exacerbation of old trouble, whereas those with an active discharge will contaminate the water and lay themselves open to the risk of added virulent infection and acute symptoms supervening on the chronic. A successful radical operation after one year may place the patient in class 1.

Patients with acute or chronic eczema of the meatus or recurrent attacks of furunculosis of the meatus are best advised not to bathe. A notice is posted in some clinics warning patients to consult their doctor about the advisability of bathing.

M.

#### **Tonsils and Adenoids: Their Medical and Surgical Aspects.**

*J. Arnold Jones, Brit. Med. J., 2:337-340, August 24, 1929.*

In a paper read before the annual meeting of the British Medical Association, held in Manchester last July, the writer claimed that there was no medical topic which was discussed with more frequency among the laity and with more nerve and ignorance than topics relating to tonsils and adenoids. The function of the tonsils is still in dispute, but it seems most reasonable to regard them as organs for the defense of the respiratory and digestive tracts during the early years of child-

hood. When a tonsil becomes chronically diseased or hypertrophied it loses its defensive powers and comes to resemble a choked filter. This is borne out by the greater frequency with which children possessing such tonsils contract infectious diseases, and the severity of the ear and throat complications in such cases.

The symptoms of adenoids could be classed in four groups: (1) Nasal obstruction; (2) Secondary affections, aural, cervical adenitis, defective pulmonary expansion, chronic bronchitis, gastrointestinal catarrh; (3) Reflex neuroses, enuresis, night terrors, twitchings, asthma, chorea, stammering and convulsions, etc.; (4) Constitutional changes, physical and mental, such as high arched palate, etc.

The diagnosis of adenoids is usually quite easy and the treatment is surgical removal. True secondary hemorrhages from adenoidectomy are rare. As to the recurrence of adenoids, Erwin Moore, in his excellent book of last year, reports a recurrence of adenoids in 10 per cent of cases, even when well removed.

The indications for tonsillectomy are: (1) Interference with speech or respiration; (2) Chronic enlargement of the cervical glands, which might be due either to chronic sepsis of the tonsils or tuberculosis; (3) Recurring sore throats or peritonsillar abscess; (4) Systemic infections attributable to a focus in the tonsil, such as rheumatism, chronic ill health, and occasionally a rare toxemia; (5) Diphtheria carriers when the infection is persistent.

The size of the tonsil is no guide to the degree of sepsis. Two important diagnostic points are: The demonstration of pus in the tonsil by pressure with the tongue spatula and the enlargement of the glands at the angle of the jaw.

Opinion is divided as to the advantage of the guillotine over the dissection method for the removal of tonsils, but the writer prefers the guillotine method.

In the discussion which followed this paper, a resolution was passed by the meeting to the effect that removal of tonsils and adenoids in children should not be done as an outpatient operation and that provision should be made for them to be kept under observation for at least 48 hours.

M.

**Anatomy and Physiology of the Vestibular Nerve (Estado actual de la anatomía y fisiología del nervio vestibular).**

*R. Lorente de Nó (Madrid), Rev. Españ. y Amer. de Lar. Ot. Rin., March-September, 1929.*

These very important studies, made at Upsala, and Madrid (under Ramon y Cajal), are based upon serial sections from mouse embryos, while most of the previous work had been made on birds and lower forms. Conclusions thus far published are as follows:

In the ampullar cristæ, a zone of diffuse innervation is common to both sides of each crista, while there are two zones of individualized innervation, one for each side. The common zone is composed of fine fibrils, the individualized lateral zones of gray fibers.

The macula of the utricle has three zones differently innervated: (a) small, calyx-like networks; (b) large networks without an intraepithelial plexus; (c) networks associated with an intraepithelial plexus. Zone *b* resembles the individualized zones of the cristæ.

The macula of the saccule has no such delicately differentiated structure as that of the utricle; the anterior part is more delicate than the posterior.

Confirming Voit's schema, Lorente de Nó has, in embryos of appropriate age, traced back the vestibular and cochlear fibers into the ganglion of Scarpa, where five regions are distinguished:

The anterior large celled region has cells corresponding to the anterior and external ampullæ and to the *b* zone of the utricle;

The posterior large celled region (zone *a*) contains cells from the posterior ampulla;

The anterior small celled region connects with the macula of the utricle; while the macula of the saccule is localized in the zone *b* of the posterior large celled region as well as in the posterior small celled region.

The separation of the cells corresponding to the three ampullæ in the ganglion does not appear in the vestibular trunk, for the fibers reunite on leaving the ganglion, reaching the medulla together.

The cochlear nerve is entirely independent of the vestibular, and the so-called anastomotic branch of Oort is an anomalous cochlear branch which remains attached to the vestibular for only a short distance. A group of nerve cells belonging to the ventral ganglion is to be found in the trunk of the cochlear nerve near the internal auditory meatus.

Study of the order of myelinization of the peripheral part of the acoustic nerve discloses that this process begins with the cristæ of the semicircular canals, and in the regions of gray fibers in the maculæ. Complete myelinization of these groups precedes that of other fibers from the same regions. Myelinization of the cochlear fibers considerably precedes that of the vestibular connections.

This work is of high importance in the solution of problems relating to the vestibular reflexes, and its publication will be continued in Prof. Tapia's excellent journal. F.

**Large Cell at the Petrous Tip; Gradenigo Syndrome. (Grande cellule pneumatique du rocher, etc.).**

*P. Mangabeira-Albernaz (Campinas, Brazil), Arch. Int. Lar., 8:1035, Nov., 1929.*

The author discovered a cell in the petrous tip 29 mm. long, 9 mm. wide and 14 mm. high. Above, it reached the Gasserian ganglion, in front the carotid canal, outwardly passed above the internal auditory meatus and was related to the supralabyrinthine cells described by Mouret. A splendidly documented analysis of recent work on the Gradenigo syndrome convinces him of the very great importance of this cellular channel of infection, and of the value of radiography of the petrous tips by the chin-vertex route in all such cases. F.

**Gradenigo Syndrome Caused by Foreign Body. (Syndrome de Gradenigo par corps étranger.)**

*P. Mangabeira-Albernaz (Campinas, Brazil), Arch. Int. Lar., 8:1051, Nov., 1929.*

The same author reports a patient who had shot himself in the right ear two years before, presenting right sixth and seventh paralysis, severe trigeminal pain, deafness and a deep granulating wound tract into the petrous, with much thick purulent exudate. Pain had only recently become manifest. There were no signs of meningitis. By chin-vertex radio-

graphs, the ball was found lying anteroposteriorly at the petrous tip, but could not be extracted by the usual Gasserian ganglion route. The facial paralysis cleared up somewhat after this operation. A month later severe pain and febrile reaction made a radical mastoid approach necessary; but even with an electric trephine the cavernous sinus and carotid were so close to the foreign body as to make extraction impossible. Autopsy disclosed the bullet lying in the groove of the cavernous sinus, which was otherwise entirely normal. A very large abscess with thick walls and distinct pedicle extended mushroomlike up, forward and back, along the hippocampus. The petrous was extremely hard throughout. F.

**Interpretation of Vestibular Reactions. (Un essai d'interprétation des reactions vestibulaires.)**

C. O. Nylén (Stockholm), *Acta Otolar.*, 13:302, 1929.

Pointing out lacunæ in the explanations offered by Bárány, Magnus and deKleijn, and Quix, all based upon the various theories of "centrifugal" or "pressure" action upon the cristæ and maculæ, Nylén details results of his introduction of amalgam into the round window, with resultant perilymphatic hemorrhages and typical nystagmus of position. He believes that such perilymphatic exudates in the region of the utricle and saccule will explain most cases of nystagmus of position, without necessarily having any lesion whatever of the perceptive mechanisms within the vestibule. F.

**Dropsy of the Labyrinth. (L'hydropisie du labyrinthe.)**

P. Závíska (Bratislava, Czechoslovakia), *Otolar. Slav.*, 1:113, 1929.

Labyrinthine dropsy is divided, following Wittmaack, into that originating from the tympanic cavity, from the meninges and from the blood. Nine cases of unilateral otitis media in which symptoms of otitis interna appeared were cleared up by pilocarpin. These were unoperated cases and must be distinguished from those of acute labyrinthine edema due to tight packing or heavy exudate following radical surgery. Závíska discusses five cases of acute labyrinthine dropsy in the course of meningeal disorders. These all cleared up with lumbar punctures and the use of pilocarpin. Following Steurer's theory of deafmutism as due to destruction of nerve tissue by



acute labyrinthine dropsy in childhood, he gives histologic data on degenerative changes found in a seven months' infant with hydrocephalus. Diabetes and chronic infections, such as osteomyelitis, are responsible for the blood borne type of dropsical irritation. By experimental work with rabbits, he was able to increase greatly the endolymphatic pressure and thus cause destruction of nerve endings. Pilocarpin, while useless in auditory nerve disturbances, will in therapeutic doses act solely upon the endolymphatic spaces of the vestibular system, promptly stimulating the elimination of toxic products. F.

**Anesthesia in Tonsil and Adenoid Surgery in France. (L'anesthésie ou la non-anesthésie, etc.)**

*Prof. J. Terracol (Montpellier), Vie Médicale, 10:1105, Nov., 1929.*

Thirty leading otolaryngologists of France hold the following opinions regarding juvenile anesthesia for adenoid or tonsil work: No anesthetic for babies or children under three years; ethyl chloride for older children; local anesthesia for adults. F.

## Books Received.

### **Proceedings of the First International Oto-Rhino-Laryngologic Congress.**

Professor Schmiegelow and Dr. Blegvad have at last completed the colossal task of editing the polyglot Proceedings of the First International Oto-Rhino-Laryngologic Congress (1928). The volume, of almost a thousand pages, is admirably printed and illustrated, and is of special value because of the discussions, in which, as usual, our Germanic friends maintain somewhat dogmatic positions. It seems unfortunate that Denmark refused, out of politeness, to permit any papers to appear; but we must applaud the laborious care which Denmark has lavished on the work of other nations. Only seventeen of some two hundred contributions are in English; the others are about equally French and German, with a number in Italian. Comparatively few papers are published with lists of authorities. In general, however, the contributions represent the finest work being done at present throughout the world. F.

### **Otologic Surgery.**

By Samuel J. Kopetzky, M. D., New York. Professor of Otology, Polyclinic Medical School and Hospital, New York. Cloth. 8vo of 553 pages with 104 illustrations, including 4 color plates; 21 charts. New York, Paul B. Hoeber, Inc., Second Edition Revised, 1929. Price, \$8.00.

It is an unusual tribute to the merit of this well-known work that a second edition has appeared within four years of the first. This publication is essentially a reprint of the former volume, which has been brought up to date in respect to surgical procedures and laboratory methods. P.

### **Handbook of Otolaryngology.**

By Professors Denker and Kähler. Paper. 8vo of 1382 pages. Berlin, Julius Springer, 1929.

The fifth volume of Denker and Kähler's monumental *Handbuch der Hals-Nasen-und Ohrenheilkunde* reaches nearly fourteen hundred pages, including general and industrial diseases, tumors of the respiratory organs, mouth and hypopharynx, diseases of salivary glands, nervous disorders, cosmetic operations, vocal disturbances, life insurance and medicolegal

considerations, and historical and recent data on teaching of the specialty. Colored pathologic illustrations and operative diagrams are of the highest class. Especially noteworthy are Denker's article on malignancies of the nose and sinuses; Thost's on benign and Kähler's on malignant laryngeal growths; Lexer's contribution on nasal cosmetic surgery; and various articles on vocal re-education after paralyses or laryngeal operations.

While the viewpoint of contributors is naturally mid-European, excellent bibliographies have brought the world's literature down to 1929 in each division of the subject. F.

**Radium in General Practice.**

By A. James Larkin, B. Sc., M. D., D. N. B., Radium Consultant on Staffs of Wesley Memorial, German Evangelical Deaconess, John B. Murphy, Washington Park Community Hospitals, Chicago, Instructor in Dermatology, Northwestern University Medical College. Cloth. 8vo of 304 pages with 28 illustrations. New York, Paul B. Hoeber, Inc., 1929. Price, \$6.00.

This is a monograph, unusually concise and complete. The subject matter, divided into five parts: I. General Considerations; II. General Diseases; III. Gynecological Diseases; IV. Miscellaneous Tumors and Carcinomata; V. Miscellaneous and Skin Lesions, is carefully prepared and the technical data are presented in a very practical manner. Each minor subdivision is followed by a comprehensive bibliography. One of the most satisfying works that has come to our attention. P.

**Surgical Technic in Oto-Rhino-Laryngology.**

**Fourth Volume—The Larynx and Esophagus.**

By E. J. Moure, Honorary Professor of the Faculty of Medicine at Bordeaux; G. Liebault, Otolaryngologist of L'Hospital de la Glaciere; and G. Canuyt, Clinical Professor of Otolaryngology of the Faculty of Medicine at Strasbourg. Paper. 8vo of 363 pages with 249 illustrations. Paris, Gaston Doin and Cie, 1930.

This last of a series on the surgery of otolaryngology completes a group of four. The first three volumes previously published dealt with (1) the ear and its related structures; (2) the nasal sinuses, the nasal chambers and the nasopharynx; (3) the pharynx and hypopharynx.

The work is presented in great detail and profusely illustrated.

## MEETINGS.

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The annual meeting of the American Laryngological Association will be held at the New Ocean House, Swampscott, Massachusetts, May 22nd, 23rd and 24th, 1930.

The annual meeting of the American Laryngological, Rhinological and Otolological Society will be held at the Hotel Chelsea, Atlantic City, May 28th, 29th and 30th, 1930.

The annual meeting of the American Bronchoscopic Society will be held at the Hotel Chelsea, Atlantic City, Tuesday, May 27th, 1930.

The annual meeting of the American Otological Society will be held at the New Ocean House, Swampscott, Massachusetts, May 19th and 20th, 1930.

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## THE AMERICAN BOARD OF OTOLARYNGOLOGY.

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In 1930, examinations will be held in Detroit, June 23rd, during the session of the A. M. A., and in Chicago, October 27th, the day prior to the meeting of the American Academy of Ophthalmology and Otolaryngology.

Prospective applicants for certificate should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Bldg., Omaha, Neb., for proper application blanks.

## Society Proceedings.

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### CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

*Meeting Held Monday, October 7, 1929.*

THE PRESIDENT, DR. SAMUEL SALINGER, PRESIDING.

#### PRESENTATION OF PATIENTS.

##### **Rhinophyma.**

DR. SAMUEL SALINGER presented a man on whom he had operated for a large rhinophyma. The interesting feature of the case to him was the unusually large development of the mass, which hung in a lobulated fashion over the tip and over the right wing of the nose down to the border of the lip. The result obtained was exceedingly gratifying, particularly in view of the fact that no epithelial grafts were required.

The amount of bleeding at the operation was inordinate and caused considerable difficulty. It was necessary to keep a tight bandage over the nose for three days before the excessive venous hemorrhage could be controlled.

#### DISCUSSION.

DR. BOOT suggested the use of the radio knife as a means of controlling the hemorrhage.

DR. BECK recalled a similar case which he had operated upon at the County Hospital some years ago, which was also characterized by huge pendulous masses. In his case considerable scarring resulted in spite of epithelial grafts.

For the control of venous hemorrhage, Dr. Beck found the application of the positive galvanic pole to be very efficacious.

DR. SAMUEL SALINGER presented a man, aged 62 years, on whom he had done a laryngectomy. He was first seen on March 22, 1929, suffering with a marked dyspnea. The larynx was almost completely filled with an infiltrating mass involving both ventricular bands and vocal cords. There was also edema

of the arytenoid and its folds. Tracheotomy was done, as well as a biopsy, which revealed a squamous cell carcinoma.

The larynx was removed on May 31, 1929, under scopolamin, morphin and local anesthesia. The carcinoma had invaded the thyroid cartilage and extended to the isthmus of the thyroid and pretracheal muscles. These tissues were removed with the larynx, also a considerable portion of the esophageal wall which was adherent to the larynx. The feeding tube was removed at the end of three weeks and the patient was able to swallow without difficulty.

One month after the operation there was a recurrence in the scar. The patient received three radium treatments over a period of three weeks, totaling about 4,000 mg. hours. There was quite a reaction which, however, subsided and the result was apparently a cure. The tracheal fistula was in excellent condition. The patient had gained about thirty pounds in weight.

The larynx on section showed the carcinoma to be unusually extensive. It formed a mass which completely blocked its lumen from the level of the bands down to the cricoid.

The case was interesting because of the extent of the growth and of the good primary result. It offers hope for the many cases that at first blush would seem to discourage surgery.

#### DISCUSSION.

DR. BECK was pleased to note that this operation is being taken up and carried out more frequently than formerly. He has for years been a strong supporter of this radical means of caring for cancer of the larynx and his results have justified his faith.

DR. GALLOWAY had had some experience with diathermy in carcinoma of the larynx in cases in which at first the results were very encouraging but later proved disappointing on account of recurrence and distortion of cartilage. He has come to feel that diathermy is not as dependable in carcinoma of the larynx as it is in other regions about the mouth and throat and was willing to admit that the operation of laryngectomy would still have to be accepted as the preferable means of treatment in all except perhaps very limited growths of one cord.

**"Legendary Lore of Otolaryngology."**

DR. ROBERT SONNENSCHNIG read a paper entitled "Legendary Lore of Otolaryngology."

(ABSTRACT.)

The love of the mystic and the supernatural is a trait of man. In all ages and among all people the use of magic, charms, incantations and amulets was rife. This is not to be wondered at when every tree, rock and glade was identified with some spirit, either good or evil. Is it strange that these people believed that sickness was the result of malevolence on the part of these beings of the unseen world who should and must be propitiated? The average person naturally believed in magic when leaders of thought fully believed in it.

Until comparatively recent times very little was known of the internal anatomy and nervous connections and functions of the ear, nose and throat. Of the internal ear and its nervous association with the vestibular apparatus there was no conception. The thyroid gland was unknown, the nose was merely a facial feature and the organ of olfaction. With reference to the ear, there are many allusions in myth and folklore. Mythologic legends tell us that Pallas Athene, the daughter of Zeus, was born from her father and sprung fully armed from his ear. The literature has a great many references to intense antipathy towards certain sounds. There is an instance of a gentleman of the court of Ferdinand who had epistaxis when he heard a cat mew. On the other hand, Galen and Plutarch and many others assigned a positive value to music as a therapeutic agent in such diseases as phthisis, gout and hydrophobia.

The nose apparently does not figure so much in mythology but we do find it mentioned in the classics. Dan Mackenzie says: "Evidence of olfactory influences is encountered in folklore not infrequently, particularly in connection with primitive medicine, and survivals of old olfactory methods of treatment are still extant, not only in the doings of the wise women of our own country and villages but also in modern scientific medicine. Of these, treatment by fumigation is perhaps the most widely prevalent, and probably the earliest method of 'smoking' was merely the replacing of an offensive odor by a pleasant one. Behind this rationale perhaps there lies the idea

of association with death of a fetid decomposit, and the expectation that a pleasant aromatic odor will naturally obviate the tendency to death."

In the olden times sneezing was regarded as a good omen and was considered as a sacred sign by almost all ancient peoples. It probably was connected with the idea that the soul was in the head and might escape.

The throat and respiratory passages in general, and the organs included in them, claim a fair amount of attention in ancient history and folklore. As one of the two most important functions of the larynx we consider the voice, which naturally figures greatly in affairs of mankind at all times. In the Scriptures God is practically represented as a voice in his communications with man. Adam heard the voice of God speaking to him in Eden, and Moses heard the voice of God when the Ten Commandments were given. But apart from divine inspiration the human voice has always had a superlative influence on the actions of men. Who does not recall the Phillippics of Demosthenes and the fiery orations of Themistocles resulting in the victory of Salamis and the crushing of Persian power? If throughout history the pen has been mightier than the sword, the voice in turn has been more powerful than the pen.

Some of the folklore remedies for disease of the throat and neck are very quaint. Harriet Alexander states that in Essex, England, it was believed that a surviving twin had the power of curing thrush in a girl by blowing three times in her mouth. In many countries the surviving twin was considered to possess peculiar curative powers in various maladies.

McKenzie states that in Lanarkshire, Scotland, as well as in England, Switzerland and Germany, a favorite method of restricting the growth of an enlarging neck (thyroid) was to bind a silk thread, preferably red, three times around it. Kanner states that the natives of Senegal produce artificial prognathus by removing the deciduous teeth (incisors) at an early age and applying constant pressure on the erupting permanent teeth, thus not only altering the position of the teeth but of the whole bony structure and formation of the face.

While it is difficult to find the "grain of truth in a heap of chaff," let us remember that the imagination of yesterday often



leads to practical application today. We should emphasize the general principle that treatment of symptoms is fallacious. Careful history taking, thorough examination, and the application of reasonable judgment lead us to the correct diagnosis. That established, one knows in most instances the prognosis and can establish proper treatment.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL  
SOCIETY.

*Meeting Held Monday, November 4, 1929.*

THE VICE-PRESIDENT, DR. THOMAS C. GALLOWAY, PRESIDING.

PRESENTATION OF PATIENTS.

**Hemangiectatic Tumor of the Tympanum.**

Presented by DR. FRANCIS LEDERER.

(ABSTRACT.)

Mrs. F. A. Z., aged 53, was first seen on August 23, 1929. She complained of (1) a pulsation in the right side of the head, (2) pain in the neck and head of about two years' duration, (3) impaired hearing of three years' duration. Her symptoms had become progressively worse and included a sort of quivering, jerky sensation in the face. She stated that several doctors had opened what they termed a "blood blister," and each time a severe and almost uncontrollable hemorrhage followed. Her general and family history were negative, as were the laboratory and physical findings. She had never had a discharging ear.

Examination of the right ear showed a large, purplish, granulomatous appearing mass in the canal on the posterior inferior wall, anterior to the tympanic membrane. This mass was quite firm and did not pulsate. There was, however, visible vascular pulsation in the region of the attic. On auscultation with a diagnostic tube, one end in the patient's ear and the other in the examiner's ear, a definite bruit, that corresponded to the patient's pulse as well as to the rhythm of her subjective pulsation, could be heard. The tuning fork tests indicated only a marked middle ear involvement; the whispered voice was heard at contact.

Roentgenologic examination revealed a "possible rarified area in the right mastoid."

Examination of the eye by Dr. Emil Deutsch showed a marked blepharospasm of the oscillating type involving the upper lid, not the eyebrow. The spasm was more marked on the lower lid, extending to practically the entire right lower

two-thirds of the face, the upper and lower lid being drawn outward during the spasm. The patient stated that these spasms were periodic, lasting from five to twenty minutes, and perhaps not recurring for hours. The right palpebral fissure was wider than the left, being 8 mm., and the right 15 mm. There seemed to be a slight proptosis of the right eye. Measured with Hertel's exophthalmeter, the right eye was fourteen, the left thirteen. The extrinsic muscles were normal. Media and other findings were negative.

The fundus examination showed no pathology. Vision, with the correction, was 20/20.

A tentative diagnosis of angioma of the tympanic membrane was made, and Dr. Joseph C. Beck was called in consultation. He concurred in the diagnosis and advised an exploratory operation. On September 7, 1928, under general anesthesia, Dr. Lederer carried out the technic of a radical mastoid operation until he reached the posterior bony canal wall, where considerable bleeding from the mass in the canal was encountered. The mastoid cells were normal but the fluid contained in each was seen to pulsate. As the posterior canal wall was taken down at the bridge a soft mass was encountered in the middle ear, which bled so profusely that the cavity was immediately packed. The dura of the middle fossa was exposed in order to ascertain the origin of the mass, and there also profuse pulsatory bleeding was encountered. In order to eliminate packing, a plastic operation on the canal was done according to his usual technic.

The histologic appearance of the tissue removed was that of a cavernous angioma. It was impossible to remove extensive portions because of the uncontrollable bleeding, but Dr. Lederer's feeling was that he was dealing with a hematoma. According to the history, this was not a new formation, and fitted in with a congenital malformation similar to a vascular nevus.

On the fifth postoperative day the patient developed a facial palsy on the operated side. Any attempt to remove the pack caused profuse bleeding, and not until the tenth postoperative day was it possible to substitute a smaller pack. The facial palsy gradually cleared up and the patient left the hospital but was observed at frequent intervals. She was relieved of

the marked pulsation, but the tendency to bleed persisted to such an extent that epidermization was permitted to occur just inside the meatus of the external canal, in order to protect the tumor mass.

At the time of the report the twitching of the face persisted and the pulsatory bruit could still be elicited with the diagnostic tube.

#### DISCUSSION.

DR. OTTO STEIN said that he had seen this patient before and was impressed by the dramatic aspect that a case of this type presented from the diagnostic standpoint. The appearance of an ear of this kind is often that of a blue drum membrane, and is mistaken for fluid within the middle ear. If one is not careful in the diagnosis he may incise a mass of this kind and meet with alarming and possibly uncontrollable hemorrhage. A blue drum membrane is often difficult to diagnose. It is sometimes due to fluid in the cavity and sometimes to bulging of the jugular. The type of case presented by Dr. Lederer did not offer this difficulty, but one should be careful not to attempt incision.

DR. ROBERT H. GOOD was reminded of a case which was under his care several years ago. The case was one of mastoid infection, with necrosis in the region of the antrum and of the tegmen. He packed the case because of the severe hemorrhage, and about fifteen hours later the nurse reported that the patient was paralyzed in one leg and one arm. The blood had gone extradurally into the temporal region. He quickly did a decompression under anesthesia, which relieved the paralysis, but the patient died from uncontrollable hemorrhage. He did not know whether the case was one of angioma or not, but there was necrosis of all the bony tissue in the tegmen.

DR. LEDERER, in closing, said that he attempted coagulation by surgical diathermy, but every time the bleeding was stronger. This patient also had suffered several spontaneous hemorrhages.

The question was one of etiology. Whether it occurred from gravity or whether it was a telangiectasis he did not know, but the condition was very annoying for both the patient and the doctor. The one thing that operation did in this instance

was to insure the patient against spontaneous hemorrhage, because there was now a web of epidermis across the canal which protected the bluish hematoma which could still be seen.

**How Physician and Social Worker Unite to Solve the Modern Hearing Problem.**

Presented by DR. WENDELL C. PHILLIPS.

(ABSTRACT.)

DR. WENDELL C. PHILLIPS of New York read an address on "How the Physician and Social Worker Unite to Solve the Modern Hearing Problem." He spoke of the origin of the efforts at rehabilitation which began before the World War, and called attention to two categories of the disabled because of deafness—one where the defect was stationary and the other progressive. He mentioned 36 states where vocational training is provided for these people through bureaus. The otologist finds a mechanism for the rehabilitation of the deafened in the American Federation of Organizations for the Hard of Hearing, which embraces 70 organized groups in the United States and Canada. These organizations are developing a personnel, almost all of whom are deafened, of social workers for this special work. None of the organizations include work for the congenitally deaf, but they are in cordial agreement with the objectives of such societies as the American Federation, to promote the teaching of speech to the deaf.

He pointed out that the Federation had adopted a program of public education and information, aiming to check the development of progressive deafness; and it is also promoting and undertaking surveys of schools to discover incipient hearing impairments. Twelve per cent of the elementary school children were found to have some defect in hearing. Dr. Phillips looks with confidence to the future in this program to check the development of serious deafness.

**Mutual Relationship Between the Otologist and the League for the Hard of Hearing.**

By HAROLD HAYS, M. D.,  
NEW YORK.

(ABSTRACT)

The speaker said that for fourteen years he had been vitally interested in this subject, starting when very few otologists

realized the social and economic handicap under which the deafened person lives. In 1912, working at the New York Eye and Ear Infirmary, he found that the clinics were so crowded that it was impossible to individualize patients, but he tried to inquire as to what the deafened individuals did and what happiness they got out of life. In 1913, he read a paper on the social and economic status of the deafened, and found that medical men were interested but did not know what to do. The result was that the New York League for the Hard of Hearing was formed, with the alumna association of Mr. Edward Witchie's school. The league gradually gained in momentum, and after the war they were able to engage the enthusiasm of Dr. Phillips, and in 1921 the federation of organizations for the hard of hearing was started. There was some suspicion at first as to what this organization was to do, but during that year Dr. Phillips and Dr. Hays formulated a constitution and by-laws, enlisted the cooperation of the American Medical Association, and secured the assistance of a number of otologists. The league could never succeed in any community without the cooperation of the otologists in that community.

There are two things to consider: First, how much assistance the otologists can give to the league, and second, how much the league can give to them. The profession gets far more benefit from the league than they ever can give to it, if they find out what is being done and how they can cooperate. In New York City the otologists find many hundreds of patients come for whom they can do nothing medically. The usual statement made by the average otologist to these individuals is either that they have otosclerosis or nothing can be done for them, but probably not one case out of 1,000 is otosclerosis. Or else one is in the habit of saying, "I'm sorry, but you are becoming progressively deafened and I cannot do anything." The psychologic effect is very bad when patients are told these things. They have learned that there are things that can be done, and it is the duty of otologists to do those things. If a patient has an advanced degree of deafness, and an audiogram shows that very little can be done, the patient should be informed that his deafness is of such nature that very little medically can be done, but that if he place himself

in the hands of a competent otologist he can perhaps keep him from becoming worse. Such patients should be told to have their ears tested twice a year, and assured that they can add greatly to their happiness if they will help themselves, get mental readjustment, so that they will realize that life is worth living. In New York they send such patients to the League for the Hard of Hearing to become mentally reestablished and ask them to try various hearing devices which the league has on exhibition to find out which is the best for the individual case. They are requested to find out what lip reading will do for them. They are also made to realize that there are many hundreds of little children who need help and this cannot be obtained unless they lend their cooperation.

The speaker felt that every otologist in the United States should be told of the wonderful work the American Federation of Organizations for the Hard of Hearing was doing, and they should be urged to cooperate with such an organization in their own town. It has been difficult to get otologists to realize that this work is worth while for themselves as well as for their patients. Three years ago the American Medical Association appointed a committee with two men from each state as members to study this question, mainly the prevention of deafness. No concerted effort has been made to accomplish any particular thing, but these men have been deluged with literature from the federation and from Dr. Hays, and he feels that eventually a proper interest will be awakened.

The work of the league was concerned largely with the adult hard of hearing, but this has turned, to a great extent, to the prevention of deafness in children, for 8 or 9 per cent of the children attending public schools are sufficiently deafened to need thorough examinations. Dr. Fowler has a splendid clinic in New York City. There children are investigated thoroughly from all standpoints and infections are cleared up. Dr. Fowler has not been able to get the cooperation of the doctors in New York City, and Dr. Hays realizes it is a problem they should help solve.

The phonaudiometer can be used in the public schools, so that forty children can be examined at one time and those weeded out who need more careful tests. In Detroit, there

is a school for the deafened, and an eminent otologist there has cooperated with the school since its earliest existence. Not only are hearing tests made but mental tests as well. The intelligence test will often show a mental coefficient below normal, and if the hearing of a child is improved by attending a special school and learning lip reading it frequently happens that they can be returned to their normal school at the end of a year. Many dozens of children are brought to the clinic in New York City, many of whom need instruction in lip reading and others need medical treatment. Excellent results can be obtained by careful observation and treatment. Dr. Fowler has treated over 200 children in three years. The children divide themselves into two classes, those with suppurating ear and those with catarrhal deafness. Over 60 per cent of the cases have been improved. The league has a large board of consulting otologists, and if the patient cannot afford to pay, any one of these men will make an examination and report to the league.

Dr. Hays felt that this problem must be met in a personal way, and that every member of otologic societies everywhere should be willing to cooperate with the American Federation and local league. The committee on the hard of hearing of the American Academy of Ophthalmology and Otolaryngology recently met to try to determine how to arrange for one central committee of the various national societies. They felt that if they could get the otologists of the country to work along certain definite lines the coordination of effort would accomplish a great deal.

A standardized method of testing hearing is absolutely essential. This can only be done by having a standardized instrument which all otologists will use. Such instruments are obtainable and the interchange of accurate records will be of great value.

Those who have been working along this line for several years believe that the time is coming when it will be possible to improve the condition of the deafened adult and the deafened child, and that some cases can be prevented by proper attention and some even cured. Much has been accomplished in the realm of physics, mainly by the cooperation of the Bell



Telephone laboratories in interpreting types of deafness and perfecting apparatus, but, up to the present, the medical profession has done very little. Dr. Hays sincerely hopes that the otologists will soon realize that preventing deafness is equally as important an accomplishment as operating upon a patient with a virulent mastoid infection.

## DISCUSSION.

DR. GEORGE E. SHAMBAUGH believed that most human happiness can be credited to humanitarian ideas, which are shared by most people, but very few have the ability to formulate plans to put these ideas into execution. Otologists have always desired to do all they can do for the deafened, but the first consistent effort to work out a helpful scheme for rehabilitation was accomplished by Dr. Phillips in formulating the League for the Hard of Hearing. The league is of great assistance to every otologist, who is now able to tell the patient afflicted with incurable deafness not that they have an affliction for which nothing can be done, but rather that through the league the door is opened to them for rehabilitation. The otologist has his own problems, which do not include the social work of rehabilitation, teaching lip reading, as well as the economic and social problems of these people. Dr. Shambaugh would be at a loss in practicing otology without the assistance which comes from the league. He agreed with the statement made by Dr. Hays, that most of the children who have defective hearing can be benefited, and many of them cured by treatment. The reason is that most of these children owe their defective hearing to tubotympanic trouble, a condition which can be readily relieved. An important question is, How far do these conditions in childhood predispose to the development in adult life of the condition which is known as progressive deafness, or otosclerosis? Dr. Shambaugh was of the opinion that progressive deafness of adults is not the sequel of the tubotympanic processes so common in childhood. Otosclerosis rarely begins during the first decade. The number of individuals suffering from deafness, as the result of otosclerosis, is very considerable. He was convinced that it is not the rare condition which early otologists believed it was, and this view of the incidence of otosclerosis

is today shared by those who have been most interested in this problem. He thought that Dr. Hays touched upon a very important question when he discussed the manner in which the matter should be presented to patients. Great tact is necessary. The facts can be told in a way which should not greatly shock the individual. It is rather the exceptional case of otosclerosis which proceeds to the point of severe or total deafness. Bezold pointed out long ago that many of these cases go for long periods apparently without any change. These facts should be pointed out to patients who are inclined to be depressed. Also, that the progress of the deafness is much more likely to be influenced by conditions of general health than by anything that is done locally to the nose, throat or ears. These people should also be warned that in all of our larger centers are to be found men—graduates of medical schools—who are willing to prey upon the helplessness of these individuals, and encourage them to allow them to carry out foolish and bizarre methods of treatment, which they should know cannot possibly influence the process that is causing their trouble.

DR. OTTO STEIN expressed his gratitude to the essayists for presenting this subject to the otologists of Chicago, for he believed the subject was not discussed sufficiently in medical societies in general. He has found that otologists suffer from a marked degree of apathy so far as the hopeless side of deafness is concerned, and seem to be quite indifferent toward making any effort to assist these patients. He has taken a great interest in the League for the Hard of Hearing, and thought that without their assistance he would often leave his patients stranded. They are greatly benefited when they become acquainted with and join the league.

Not long ago, in discussing this subject at a dinner, one of the prominent men in the city said he thought there was a tendency on the part of the league to interfere with the practice of medicine, that it rather smacked of State medicine and he was against it. Dr. Stein feared that this feeling existed with others because they were not aware of the real situation and do not trouble themselves to find out what splendid assistance the league gives.

DR. JOHN J. THEOBALD said he has been engaged in detecting the hard of hearing rather than in the reeducation.

He has examined with the whispered test about 30,000 school children under the auspices of the Chicago League and found about 3 per cent to have definite hearing defects. Considering that there are about 500,000 school children in Chicago and that it has taken six or seven years to examine 30,000, it is apparent much remains to be done. The examinations, while slow, were rapid enough to keep pace with the follow-up work. They did not have the advantage of an audiometer until the last school was examined recently. At this school they used the 4 A audiometer and also the whisper test method. Comparative results will be published soon. Audiometer reports vary from 2 to 16 per cent. This variation is too great and there is urgent need for standardization. They have to be very careful to avoid such high percentages by rechecking, for when children not clinically hard of hearing are sent to clinics or to their physicians they are often returned with a negative diagnosis and this discredits the work, for parents want to hear that their child is normal. With the whisper test and tuning forks they found about 52 per cent of the cases tubotympanic catarrh, which was greatly benefited by inflation. Advanced cases of catarrh which could not be benefited by inflation were advised to learn lip reading. Dr. Theobald thought it would be an excellent thing if children could be taught part time lip reading as an adjunct to their other school work, where the present defect is not sufficient to warrant full time lip reading instruction. Lip reading instruction should be urged in all cases in which there is a progressive form of deafness.

This group includes, besides chronic catarrhal cases, bilateral chronic suppurative otitis media, otosclerosis and some cases of nerve degeneration. One must not be misled by the apparently fair hearing ability in a child suffering with the above progressive conditions. One must think rather of the almost inevitable loss of hearing over the ensuing period of ten to twenty years despite all forms of treatment.

They have not yet been able in the past to arouse much enthusiasm in Chicago in regard to introducing routine examination in the schools, although since the advent of the 4 A audiometer there are definite signs of activity. He had recently been informed that the Chicago school system contem-

plated getting three of these instruments and going into the examinations on a large scale.

This was very gratifying news to the Chicago League for the Hard of Hearing and to the medical profession in general, as it is the beginning of the solution of an old problem.

The profession is indebted to Dr. Phillips and Dr. Hays, who have been directly responsible for the formation of the leagues for the hard of hearing which, in turn, have provided the stimulus for routine examination of school children.

DR. J. HOLINGER commended the splendid work being done by the League for the Hard of Hearing in Chicago, and stressed the great responsibility of the doctor who handles these patients. Many of them become desperate and attempt suicide when they are informed that they have progressive deafness and that nothing can be done for them. It is not the otologist alone who should be informed about this work, but the general practitioner who sees the patients in the early stages of deafness, and often sees nothing for them beyond advising the use of "sprays" for the nose. If they get a better understanding of these cases and realize the seriousness of delaying proper diagnosis and advice, much more can be accomplished for the patients. Most of the doctors do not realize that they are neglecting these patients cruelly.

He felt that Dr. Hays must be mistaken when he said that not in one out of every thousand cases in which the diagnosis of otosclerosis was made by otologists this diagnosis was correct and could be verified by pathology. Dr. Holinger insisted that the opposite is true, namely, that in every case where the diagnosis of otosclerosis was made in the living according to the teachings put down by Bezold and Siebenmann, the characteristic changes were found in the dead. Before Siebenmann cleared up the situation it was believed that a large part of the disease was due to the changes in the blood vessels, because of the loud thumping noise synchronous with the pulse. Therefore Siebenmann worked out his beautiful atlas of the blood vessels of the labyrinth.

DR. ALFRED LEWY asked if either of these gentlemen knew of an electric device in this country that is especially fitted for inner ear deafness. He believed that most of the amplifiers

as now made are unsuitable for this condition. Professor Flatau has presented an instrument before the German Otolaryngological Society that is alleged to damp lower tones and amplify higher ones and be of aid in inner ear deafness.

DR. G. HENRY MUNDT thought the question of sending these patients to physicians was very interesting and brought up a point which might well be considered. There is a strong tendency among many medical men to agree with parents that there is nothing wrong with their child. He believed it might be possible to prepare a statement that could be printed on the back of the examination blank stating that the child had shown a certain percentage of deafness. The same thing applies with deafness as with strabismus in young children, but it is difficult to make people understand that these things should be cared for during early childhood. Dr. Mundt has had an unusual opportunity for observing these children, for he lived next door to a school for the deaf for several years. He believed that only a person who has seen these children develop can really understand what they get and what they can get out of life. No otologist can follow his deafened patients unless he makes audiograms frequently.

The League for the Hard of Hearing he considered the type of organization that the medical profession would be pleased to work with, and suggested that some effort be made by the society to broadcast to the general practitioners something of the scope of the work and the opportunity to really serve these patients. Several times a year he sees young children who are undoubtedly deaf, and those are the cases he spends the most time with. He talks to the parents and makes them understand that the child should be placed at once where its education can be started, instead of waiting until it is grown or half grown, as so many practitioners advise.

DR. AUSTIN A. HAYDEN expressed his appreciation of the presentation of this subject and his interest in the discussion. He hoped that the habit of willing temporal bones to physicians would increase, particularly if the bones were accompanied with a detailed history of the case from its inception. Not until a number of such cases have been obtained can a comprehensive study be made and recorded.

Dr. Hayden felt that the discussion of the papers could be summed up in two things: First, the otologists should cooperate with the League for the Hard of Hearing. If the problems of the deaf and the deafened are to be solved by the proper people the medical profession must contribute a large share of the effort and must guide the work and interests of these individuals into proper channels or great harm will be done to the deafened today, and a great opportunity will be taken away from those who are to come. The society owes a great debt of gratitude to its Council of last year in the fact that the Council adopted a resolution by which every member of the society was automatically made a member of the Chicago League for the Hard of Hearing. Dr. Hayden believed this plan should be put in force all over the country, so that each otologist would become an integral part of the leagues for the hard of hearing.

Second, he felt that otologists must come to standardize the measurement of hearing defects. He believed it would be agreed that much of the work Dr. Theobald has done, and much of the excellent work Dr. Shambaugh pioneered in, and the work Dr. Hagens had assisted with in the institutions, would have been more valuable if the tests had been made with the 4 A audiometer than with the whispered test. The same thing is true of the otologist in the office. They should use not only the 2 A but also the 4 A audiometer, and should standardize the whisper test.

There is a great deal to be done for the deafened by the medical profession, and in return perhaps some things that they can do for the doctors. Every otologist should be vitally interested in this problem that affects such a large percentage (perhaps 10 per cent) of the population.

DR. A. M. CORWIN was impressed with the fact that every otologist should broadcast to his own conscience the fact that in the League for the Hard of Hearing we have a body of specialists who are doing work that the profession never has done and never will do, for they do not know how to do it. It is a most important thing for these handicapped individuals, and it is the duty of every physician to refer the deafened to these laboratories of the League for the Hard of Hearing.

The sooner they realize that there are laboratories with which they can cooperate, and that they are doing excellent work, the better will be the progress.

DR. WENDELL C. PHILLIPS, in closing, said that before making any attempt to educate the general practitioner as to what his duty is, the otologists of the country should be educated. Very few of them have any idea of what is needed, and he felt the members of the Chicago society could consider themselves far in advance of the general run of otologists.

Second, it is necessary to solve the problem of deafness, of whatever type it may be. He expressed himself as all for the work of the American Otological Society and the others that study temporal bones, and in full sympathy with the work among the deafened children. Some time the problem will be solved. It may be in the laboratory through the study of temporal bones, or it may be through the study of the diseases beginning with the young children. Dr. Phillips was more inclined to think through the latter.

As to electric hearing aids, there are certain deafened persons who never can hear with any electric device. Very old persons who are almost totally deaf very rarely can hear through any such device. He has seen very few cases of absolute deafness, but recalled one hopeless case in which the patient was totally unable to hear any sound whatever.

DR. HAROLD HAYS, in closing, expressed his appreciation of Dr. Shambaugh's remarks and of his sympathy with the work they are doing.

He agreed that although many cases have proved to be otosclerosis by laboratory investigation, many individuals are down and out because this diagnosis has been given them when it was later proved that the deafness was due to something entirely different. The greatest bugbear today is this diagnosis. Patients are afraid of the term otosclerosis, and they do not believe, as Dr. Shambaugh does, that such a condition can be improved. He stated that he did not claim to know any more about otology than anybody else, but he feels that many cases have been diagnosed as otosclerosis which showed a deafness due to a relaxation of the ear drums or to some systemic toxic factor.

**Meningitis (*Staphylococcus Aureus*) Secondary to Sinusitis With Operation and Recovery.**

BY DR. HOWARD C. BALLENGER.

(ABSTRACT.)

A number of recoveries of meningitis have been reported in the literature, but where the diagnosis of a suppurative meningitis has been substantiated by the recovery of the causative organism in two or more lumbar punctures, the number of reputed recoveries is greatly lessened.

Kolmar in writing of pneumococcic and streptococcic meningitis says: "The mortality of diffuse spreading types with purulent cerebrospinal fluid is nearly 100 per cent."

Neal, in 1914, found only five cases of recovery following streptococcus meningitis. Lamar, in 1912, collected thirteen cases of cures in pneumococcus meningitis. Eagleton, in 1912, in an analysis of the literature of reported recoveries of suppurative meningitis in which the diagnosis was substantiated by finding organisms in the cerebrospinal fluid, collected only thirty-one cases (including his own). Of these thirty-one cases, six were somewhat in doubt, either of being a meningococcus, or without sufficient data to clearly prove the cases as a suppurative meningitis.

Goldstein and Goldstein, in 1927, after a review of the world literature, estimated about 150 cases of recovery from pneumococcus meningitis.

The reference in the literature to staphylococcus meningitis is rather meager, probably due to the comparative infrequency of its occurrence.

Neal, in 1924, examined a list of 1,535 cases of purulent meningitis. The frequency of the causative organisms in the order named were meningococcus, pneumococcus, streptococcus, influenza bacillus, staphylococcus and bacillus coli. Cases due to the last two organisms were found to be comparatively rare.

There seems to be a general impression that a purulent meningitis due to a streptococcus is more virulent than one due to a staphylococcus. Perusal of the literature fails to offer evidence for or against this impression. Pandey attributes more importance to the virulence of the organism than to its



type. Recoveries from staphylococcic meningitis have been reported by Dandy, Moise, Lortat and Grivot, Salvini, Emerson, Wharry and probably others.

#### RECORDS OF CHILDREN'S MEMORIAL HOSPITAL.

An examination of the records of the Children's Memorial Hospital for the years 1909 to August, 1929, of all cases of meningitis (exclusive of tubercular meningitis) reveals a total of 297 cases, of which 273 were epidemic meningitis, leaving twenty-four cases (excluding my own) of septic meningitis. All but one of the twenty-four cases of septic meningitis died in one to fifteen days. This one case (No. 2) left the hospital without permission with the child in a dying condition.

The various white blood cell counts were recorded in fourteen cases. They varied from 3,250 to 58,800, giving an average of approximately 22,000.

The cell counts of the spinal fluid taken by lumbar puncture were recorded in nineteen cases (one case marked many). They varied from 210 cells to 14,400. An average of these counts gives a figure of about 3,800.

A culture of the spinal fluid was recorded in twenty-three cases. An organism was obtained in one or more cultures during life in twenty-one cases. In the two cases in which an organism was not demonstrated during life an organism was obtained from a culture taken at the autopsy. A streptococcus was responsible in thirteen instances. A pneumococcus was found in ten cases.

A culture of the blood was recorded in five cases. In four instances a positive report was obtained. All the positive blood cultures showed the same organisms that were found in the spinal fluid.

#### REPORT OF CASE.

R. L. (2680-29), boy, 6 years of age, entered the Children's Memorial Hospital (Chicago), August 17, 1929, complaining of headache, fever, convulsions and swelling of the forehead and left eyelid.

The illness began two days before admission, with a frontal headache and a purulent discharge from the nose—worse on the left side. His nose had been discharging a mucopurulent secretion for the past month. He was said to have had sinu-

sitis. The day before admission he began to have some swelling above the bridge of the nose and the midline of the forehead, accompanied by a high fever. In the evening he began to jerk and soon to convulse generally. These convulsions lasted about two hours until relieved by packs and chloral. No vomiting occurred.

The past history was negative except that one month ago he had an attack of diarrhea and vomiting.

The physical examination revealed a well nourished child of six years who was very irritable and febrile. The ears and abdomen were negative. A diffuse, brawny, tender swelling, without redness or fluctuation, was present over the middle and lower forehead and upper part of the nose and left upper eyelid. Rigidity of the neck was present, Brudzinski absent, knee jerks not elicited, Kernig positive, Babinski not constant, ankle clonus not present.

The blood count was 16,000, 88 per cent polymorphonuclear and 12 per cent lymphocytes.

The spinal fluid was turbid and under moderate pressure with a two plus positive Pandy test. The cell count was 320 with 96 per cent polymorphonuclear and 4 per cent lymphocytes. Culture of the spinal fluid taken at this time showed gram positive staphylococci, which on further growth proved to be *staphylococcus aureus*.

The following day his condition was worse, with increased swelling and tenderness of the forehead and upper left eyelid. He was irrational at times. Positive Brudzinski and positive bilateral Kernig tests were present. Ten cc. of concentrated antimeningococcus serum were given.

The following day (August 19th) I saw the child in consultation with Doctors P. F. Morf and G. P. Weiler, who had referred the case to me. Inasmuch as his condition was growing worse and the signs of the meningitis were increasing, it was decided that he might have a slim chance if the left frontal sinus was opened externally with removal of the inner bony table to establish drainage from the probable point of entrance of the infection to the meninges. This was done the following day. The left frontal sinus was filled with a thick yellow pus. The mucous membrane lining the sinus was detached and appeared to be partly destroyed. After the inner

table of the frontal sinus was removed the same yellow thick pus escaped from the dura with a more or less continuous flow. Culture of this pus revealed staphylococcus aureus. The right frontal sinus was opened by removing the septal wall separating the two sinuses. Pus was also found in the right frontal sinus. Cigarette drains were stitched in the wound, one drain going to the right frontal sinus and another drain to the dura. The wound was not closed. Lavage of the brain was not used at the operation or subsequently. His general postoperative condition was good.

Cultures taken from the frontal sinus, from the dura, from the blood, and from the nose at various times all showed staphylococcus aureus.

Three days after the operation pneumonia developed in the left lower lobe with later involvement in the right lower axillary region. The pneumonia was probably of a metastatic or a hypostatic type.

A day or so later a profuse discharge of pus was obtained from beneath the outer portion of the upper eyelid, probably a burrowing of pus from the left frontal sinus. The spinal fluid continued to show the staphylococcic organisms (with one exception) each time a spinal puncture was made up to and including September 11. (Three weeks after the operation.)

The cell count varied from 260 to 3,350. In the instance in which the cell count was 3,350 the polymorphonuclear cells were 94 per cent and the lymphocytes 8 per cent, which was about the proportions held throughout, with a slight relative increase in the lymphocytes at times.

The highest white blood cell count was 32,400, taken August 25, five days after the operation.

This same date (September 3, 1929) two fluctuating elevations, about the size of a dollar, developed over the occipital region and over the parietofrontal region, which upon incision proved to be abscesses containing a large amount of thick yellow pus. A pure culture of staphylococcus aureus was obtained from each abscess. A few days later a third abscess occurred in the left parietal region, which also showed staphylococcus aureus. Roughened bone could be felt under each abscess. The abscesses were probably due to an osteomyelitic

process. One abscess was still draining with a diminishing discharge at the present date (November 1. 1929).

In the three instances in which blood cultures were taken, positive cultures of staphylococcus aureus were obtained twice. The first negative blood culture was found 19 days after the operation.

He was semicomatose, drowsy and irrational at times during the most of his illness, with occasional attacks of vomiting. Headaches were complained of frequently. Many times he would waken with a loud cry (meningeal cry).

September 11th, or the 21st postoperative day, he suddenly developed a complete paralysis of all muscles of the right eye with a dilated pupil which did not react to light. He could count fingers in this eye and later could read large type. There was some question whether a slight choking of the disc was present or not. The paralysis of the pupil and the muscles of the right eye had continued to date, with some improvement. However, all evidence of sepsis had disappeared. He had no other evidence of a mental or physical defect with the exception of a slight discharge from one of the scalp abscesses. (November 1st.)

In summing up, this boy presents the picture of a double frontal sinusitis with a suppurative meningitis (staphylococcus aureus), complicated by bronchopneumonia and multiple abscesses of the scalp, probably of osteomyelitic origin, with septicemia, with drainage of the brain through the probable site of the origin of the infection, with recovery.

#### DISCUSSION.

DR. J. HOLINGER asked whether there was any experience which might give information as to whether, in the early use of the serum in epidemic cerebrospinal meningitis, there might be fewer cases of subsequent total deafness. This question was prompted by an experience in the Alexian Brothers Hospital a number of years ago. Four boys of the same family were brought in with cerebrospinal meningitis. They received the serum as soon as possible. One boy died and the other three were discharged with beginning deafness. One doctor told the mother that the boys would be all right, but Dr. Holinger told her that he thought all three would be deaf, and

so it happened. He wished to know whether it was the general experience that deafness does not occur if the serum is used early, and how early it must be used.

DR. T. C. GALLOWAY expressed his interest in this report and his appreciation of the courage of Dr. Ballenger. It should make the profession appreciate the possibility of curing such a case, no matter how hopeless it might appear. He recalled three cases they had reported from the Cook County Hospital several years ago of progressive meningitis, in which they would have been justified in doing nothing, but in all three cases they got recovery.

DR. HARRY L. POLLOCK cited a somewhat similar case, in which the patient recovered without operation. The patient was a boy, aged 12, upon whom during an acute cold an ethmoid operation had been done. The boy was ill, had pus in the nose, and a doctor curetted out his ethmoids at home, with the aid of a lamp. The boy was brought to the hospital with symptoms of an ordinary meningitis. The cell count was 1,200 on the first day, and all other tests were positive. There was no evidence of an ethmoid infection except a general thickening and secretion in his nose. They shrunk up the tissue and sucked out the secretion, and twice a day did a spinal puncture, removing all the spinal fluid that would come out each time. This was done for fourteen days, and they recovered the staphylococcus in the fluid. No serum was given or anything else, only the spinal drainage. They had a neurologist see the patient and he suggested making a cisternal puncture, and nothing else except a little sedative in the beginning to quiet him. At the end of three weeks the boy recovered. He had been watched since and there had been no permanent damage.

Dr. Pollock congratulated Dr. Ballenger on his effort and on the splendid result he obtained.

#### **Sodium Iodid in Hyperesthetic Rhinitis.**

BY ALFRED LEWY, M. D.

For about two years I have been using, with considerable success in the treatment of hyperesthetic rhinitis, free iodine solution derived from sodium iodid, following the method recommended by Sternberg and Sugar (*Zeitschrift f. Hals, Nasen*

u. Ohrenheilkunde, 15-357, 1926). These gentlemen reported favorable results in acute and chronic nasal discharges and in hyperesthetic rhinitis from the use of the halogens in free solution. The technic with iodine is as follows: A 3 per cent solution of sodium iodide is prepared, sterilized and allowed to stand several days in a white glass stoppered bottle until free iodine appears. One c. c. of this solution is injected hypodermatically. There is sometimes a temporary exacerbation of the symptoms. The injection is not repeated within five days. After standing several weeks the injection becomes painful because of too much free iodine, which is also shown by the increasing yellow coloration, and the solution should be discarded and a fresh solution prepared.

If the result is favorable the injections are repeated as necessary. Most of my cases have shown recurrences, requiring occasional repetition of the treatment, but some appear to be permanently relieved. Several cases have been relieved by this method after a careful study of allergic reactions and various local treatments had yielded no results. I have had some favorable results in the initial stages of acute coryza.

In a more recent article these same authors recommend a 3/10 per cent solution of sodium iodide in patients in whom the 3 per cent aggravates or brings no results. I have tried this twice on patients in whom the 3 per cent solution was ineffective. One of these cases was promptly improved; the other was not helped. With the other halogens I have had no experience.

**Milk of Magnesia and Olive Oil as a Topical Application to  
Mucous Membranes.**

BY ALFRED LEWY, M. D.

Equal parts of milk of magnesia and olive oil have been used by dermatologists for some time for skin irritations, but I have not known of its use for acute inflammations of the nasal mucosa. For some months I have been using this combination with satisfaction to my patients and myself. There may be added to this base any of the essential oils, singly or in combination, as desired, my favorite combination being 15 or 20 drops of oil of white pine or of eucalyptol to the ounce of the above mixture, which appears to be quite stable but requires

occasional shaking. It is especially useful when discharges are excoriating. In subacute inflammations of the nasal mucosa I have used equal parts compound fluid extract of benzoin, milk of magnesia and olive oil. The above combinations appear to me to be more satisfactory than any silver salts that I have used, but have the disadvantage of not being applicable through the ordinary medicine dropper for home use. For this purpose I have used a one-dram syringe with a large opening and heavy rubber bulb. It may be that the preparation will work well in a collapsible tube.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL  
SOCIETY.

*Meeting of Monday, December 2, 1929.*

THE PRESIDENT, DR. SAMUEL SALINGER, IN THE CHAIR.

SCIENTIFIC PROGRAM.

DR. EDWIN MCGINNIS addressed the Society on "Incidental Mastoiditis."

(ABSTRACT.)

**"Incidental Mastoiditis."**

Upper respiratory infection may be a very simple affair, evidencing itself as only a simple nasal cold of about a week's duration, or it may be more serious and its progress through the body may reach into many distant regions.

Mastoid infection may be the end result of an acute coryza or tonsillitis, and if the infection is limited to one or both sides its cure may be accomplished by a simple mastoidectomy. This is a happy end result, because the patient's pain is usually relieved by the excision of the infected cells and the leucocytosis subsides, the temperature comes down to normal, and the end result is a satisfactory one.

Occasionally a patient presents who has the classic symptoms of mastoiditis, and if operation is performed there is not the happy return to normal health. An early contact with such a patient was one who had had a mild tonsillitis, then a one-sided otitis media, and later a mastoid involvement. Early myringotomy started the ear draining and the patient seemed better for a time, but soon he developed a rather high temperature, was dizzy, and then had nausea and vomiting. A diagnosis of brain abscess was made. Later a cerebellar abscess was opened and drained on the side opposite to the infected ear and mastoid, and the supposition was that the abscess had its inception in the tonsillitis, and that the mastoiditis was incidental to the primary tonsillar infection.

This incidental infection of the mastoid is more clearly illustrated by the following case in the practice of Dr. W. W. Wilson:



Miss G. G., aged 24, of Aurora, Ill., had an acute coryza, beginning about March 10, 1929. This was followed by pain in the left ear. Examination at that time by Dr. Wilson showed tympanic congestion but no bulging. The temperature was about 100° F., but the patient appeared more ill than the clinical findings indicated.

March 12, under nitrous oxid anesthesia, a red and bulging drum was opened by Dr. Wilson, and a thin greenish pus emptied out of the middle ear. Short chain streptococci were found in this pus. From March 12 until March 28, she ran the course of an acute otitis media, with the temperature not going above 101° F.; she seemed to be getting better, but on March 28 had a chill, and the temperature rose to 105° F. On this date she entered St. Charles Hospital, and on April 2, her temperature reached 107.4° F.

There was moderate tenderness over the mastoid, and the ear was draining a serosanguineous fluid. Dr. McGinnis examined her on March 30, and at that time her white cell count had dropped from 10,850, on March 29, to 6,500. On March 31, it dropped to 5,300. It was thought best to delay mastoid surgery until after a blood transfusion, which was done on April 1. At the examination on March 30, he felt sure that she was suffering from a general sepsis which had its entrance through the nasal cells, because she had painful cervical glands on both sides and appeared more ill than the mastoid findings indicated. The question of lateral sinus infection and thrombosis was discussed at this time.

April 2, Dr. Wilson and he did a simple mastoidectomy, and as the sinus wall looked perfectly normal it was left alone. The patient made an uneventful recovery from the operation, and the mastoid wound drained and healed in about four weeks.

From April 2 to April 23, she ran a septic temperature, the maximum being 107.2° F., with numerous chills. On April 15th she complained of severe pain in the left iliac region. Heat and flexion of the left thigh on the abdomen relieved the pain. A palpable and tender mass could be outlined in the left iliac region. April 23rd, under nitrous oxid anesthesia, a left muscle splitting incision was made, and a large amount of thin, bloody, purulent material drained from this region. Bacteriologic examination of this discharge showed short chain

streptococci. A rubber drainage tube was inserted and the patient made an uneventful recovery.

But this was not the end, for she continued to have fever up to 104° F., from April 23rd to May 20th.

On May 15th, the patient developed a severe pain over the right kidney region, and mild chills, not similar to those of the onset. On May 21st an incision was made along the lower rib border postperitoneally, and the perirenal space was explored. No pus was found, but the patient commenced to improve following this.

A second blood transfusion had been given on May 10th. The temperature finally became normal about May 29th and had remained normal since.

The patient was discharged from the hospital June 1st, and had made an uneventful recovery, without symptoms. She had gained upwards of thirty pounds.

His first impression on examining this patient was that she had more than a mastoid infection—in other words, general sepsis—and he thought that the ear was not the only point of entrance, because the jugular on that side was not tender on palpation, and tender and painful glands were palpable on both sides of the neck. With this in mind, they did not disturb the lateral sinus and the jugular. He still felt that ligation of the jugular would have had very little helpful effect on the progress of the infection. They also wanted to eliminate shock as much as possible. He believed that the first blood transfusion was very helpful, in that it raised her white cells and carried her through the mastoid surgery.

His impression of this case was that the mastoiditis was just an incident in the general infection, and its elimination by operation was helpful in the cure.

He expressed his appreciation to Dr. W. W. Wilson for the chance of participating in this very interesting and instructive case, in which they had the complete cooperation of the Brennecke clinic and the St. Charles Hospital.

#### DISCUSSION.

DR. C. W. HAWLEY volunteered the report of a case similar to one reported by Dr. McGinnis.

A number of years ago at the Postgraduate Hospital he was consulted by a patient suffering from a neglected mastoid involvement on the right side. He performed a mastoid operation and for a few days the patient did well, but then an extensive abscess formed in the right shoulder. This abscess was opened and drained by the surgeon who referred the patient. A week later another abscess formed in the knee joint, which was also opened with a considerable discharge of pus. A prominent surgeon was called in consultation and he said everything possible had been done. The patient continued to lose weight and strength, and Dr. Hawley suggested giving vaccine, to which the others did not agree. Within a few days there was involvement of the ankle joint. An autogenous vaccine was then prepared and 15 minims injected on each of three successive days. On the fourth day the dose was reduced to 10 minims. At the end of two weeks the abscesses had entirely healed and the one that threatened the ankle joint did not materialize.

Dr. Hawley expressed the opinion that a patient suffering from any acute infection will stand larger doses of any medicine than he can stand in the course of a mild illness. Eighteen years ago he had an acute attack of facial erysipelas in which no vaccine was used, and two years later had another, which he recovered from promptly following the use of vaccine. There was a recurrence in a few weeks, which also responded to vaccine therapy in 15 minim doses on two successive days.

He also cited a case of multiple abscesses in the neck, which improved promptly under vaccine. He believed one should not be afraid to use liberal amounts in desperate cases.

Dr. JOSEPH C. BECK thought the point for discussion was the recognition of an infected lateral sinus that looked normal. The case Dr. McGinnis had described was a multiple secondary infection, which he thought would have recovered without operation.

He recalled a case at the University Hospital some years ago in which pneumococcic symptoms were very prominent, and this germ was thought to be responsible for the septic condition of the patient. The jugular was not painful and there was nothing to indicate that thrombosis was going on, but the picture was so suggestive of a thrombophlebitic type of

mastoid that operation was determined upon and the patient made a good recovery. He thought, from the standpoint of the otologist, knowing that the lateral sinus becomes infected easily, there is no harm in opening and treating it properly. This is far better than to allow the patient to develop a septic endocarditis or a septic infarct in the lung.

DR. SAMUEL SALINGER cited a case seen in consultation with Dr. Sonnenschein. The patient was a young lady who had had a mild otitis media that had discharged for only two or three days. Although there were no local signs of mastoiditis she developed chills and fever within a period of five days with a temperature of 105 to 106 degrees F., and after eliminating all other possible foci it was decided to open the mastoid, which was found to be absolutely normal. Exploration of the sinus showed that it also was normal. Nevertheless the jugular was ligated and the patient made a prompt recovery. The case was evidently a direct blood stream infection by way of a small emissary vein, or else a mural thrombosis was present too fine to be detected. As noted above, the mastoid and the sinus were grossly normal.

DR. EDWIN MCGINNIS, closing, said he did not think the case was a lateral sinus thrombosis. He cited a case seen some years ago, in which a young lady had an infection around the lower incisors and developed an abscess that extended underneath the periosteum of the inferior maxilla. This area was opened and treated by gentle curettage and cleared up nicely. In two or three weeks she came in complaining of fever in the afternoon and pain in the left groin. Her temperature was 101° F. She was placed in the hospital and with the help of a urologist, a left-sided perinephritic abscess was diagnosed. An incision was made along the left lower border of the rib and a large amount of pus escaped. This abscess could not be determined upon percussion or palpation because so deeply situated.

Dr. McGinnis has sometimes used vaccines in cases such as Dr. Hawley described, but in the case reported it was a non-suppurative variety of bacteria. He agreed that a mixed infection could exist without much change, because in sinus infections there can be marked involvement without much visible change, and the same might hold true of the mastoid.

Dr. SAMUEL J. PEARLMAN and Dr. SAMUEL SALINGER presented a paper entitled

**"Hyperpyrexia (Fatal) Following Tonsillectomy."**

(AUTHOR'S ABSTRACT.)

Following operations, major or minor, conducted under general anesthesia, about the oral cavity or away from it, there has been noticed what may be termed a syndrome of pallor and hyperpyrexia. These symptoms come on a few hours after operative interference and occasionally result fatally, within twenty-four to forty-eight hours. Postmortem with few exceptions has thrown surprisingly meager light on the cause of death. The commonest findings appear to be cerebral congestion and mild internal and external hydrocephalus. Inasmuch as the cause of this syndrome is not known, there can be no rational preventive therapy. It would seem best not to operate on any individual, particularly a child, with a temperature above 99.2° F.

DISCUSSION.

Dr. ROBERT SONNENSCHNEN said that the public and many physicians have come to believe that tonsillectomy is a very simple and harmless procedure. The fact that so many tonsillectomies are performed throughout the country, so often by general practitioners, with few serious sequelæ, is no doubt the cause of this erroneous belief. Many operators disregard the fact that serious if not fatal hemorrhages or other postoperative results, such as infection of the throat and ears, sometimes occur, but as long as they do not result fatally, they consider them as inconsequential results. They likewise pay too little attention to possible distortion and stenosis of the pharynx, due to excessive scar formation following the removal of portions of the anterior pillar, the soft palate and uvula, to injury to the mucosa of the posterior pharyngeal wall in the removal of adenoids, etc. One thing, however, has put the fear of God in them, and that is the very unfortunate and occasional death which results from tonsillectomy.

Dr. Fetterolf, at the last meeting of the American Laryngological Association presented a very interesting case report in

which hyperpyrexia followed tonsillectomy and persisted for a number of weeks. No etiologic factor was discovered for a long time, but they finally found the bacillus abortus. This bacillus often infects cattle and produces abortion. The patient shortly afterward recovered. Dr. Fetterolf said that the best treatment for this condition is the intravenous use of mercurochrome, and expressed the opinion that in all cases of hyperpyrexia lasting for a considerable time, without other cause, this bacillus should be sought.

As demonstrated in this very interesting paper, the exact cause of death is often not ascertainable, even by autopsy. Either the changes are microscopic, so that they are not detected at the time of postmortem examination, or they are not even discernible when histologic examinations are made. Only recently Dr. D. J. Davis stated before this society that in many of the tissues decided changes take place, even a few moments after death, so that we can readily see that where postmortem examinations are delayed a number of hours or even longer, the changes in the tissues may be such that the exact pathologic process responsible for death cannot be diagnosed.

It was impossible for him to throw any light upon the unfortunate ending of some of these cases. The references show that a number of theories have been advanced. It is well known that acidosis occurs very frequently after a general anesthesia in children and is often accompanied by very high temperature. Fortunately, most of these cases respond very well to the administration of alkalies and carbohydrates, so that the patients usually recover rather promptly. Should the acidosis, however, be very severe, coma might ensue just as it does in cases of diabetes. It seemed to him that the theory of death based on the unstable nervous system in small children, together with some disturbance in the heat regulating center, may account for some of these fatalities. No blame can be attached to the operator if all reasonable precautions have been taken, such as a careful examination of the urine, of the heart, thymic region, etc., but one must ever bear in mind the possibility of grave postoperative results. Both the surgeon and the layman should recognize the fact that there is danger connected with any operation, and the mere fact that a certain type of operation

is very often performed does not obviate the possibility of very serious and wholly unexpected results. Since there are inherent dangers in all operations, none should be advised unless a thorough examination and a definite history give clear indications for surgical interference. Furthermore, all possible care in the performance of the operation and in the post-operative course is imperative. Then and then only can our consciences be clear when these inexplicable and unfortunate things are noted after a carefully performed operation.

He said the essayists had rendered a great service in bringing attention to this series of cases and for giving such an excellent exposition of the subject.

DR. J. HOLINGER said that in a recent number of the *Zeitschrift für Ohren, Nasen und Halskrankheiten* a number of similar cases were recorded. When death occurred the parts around the tonsils were very carefully dissected and it was found that in a certain proportion of the cases the large veins from the tonsils were thrombosed with septic thrombi. The thrombophlebitis progressed along the large veins to the cavernous sinus at the base of the brain. The connection from the tonsils to the sinus is short, and a thrombophlebitis in these regions is rather difficult to find. Recently reports of ligation of the veins have been published.

He did not know whether after tonsillectomy the cavity should be swabbed with one or the other of the strong antiseptics. He likes to use peroxid, which has the double advantage of stopping the hemorrhage and of being a strong antiseptic which does not injure the tissues.

DR. ROBERT SONNENSCHNIGER thought the explanation suggested by Dr. Holinger would not answer in these cases, for the first patient died within sixteen hours after operation, and no infection could produce a septicemia and cause death in that time. In the *Monatschrift für Ohrenheilkunde*, etc., Waldapfel, who read a paper before the American Academy of Ophthalmology and Otolaryngology, in 1928, had an article in which he stated that dissections have shown that probably in many cases of tonsillectomy there is some thrombosis in the veins along the tonsils, but this did not cause a pyemia.

DR. GEORGE W. BOOT said it was a well known fact that there is such a thing as heat stroke, but at postmortem often

nothing can be found to explain it. There is a septic temperature due to thrombosis of the peritonsillar plexus following tonsillectomy, and he had seen such a case at the Children's Memorial Hospital that persisted for several weeks but finally recovered.

Referring to acidosis after anesthesia, he asked if anyone had seen it following nitrous oxid.

DR. OTTO STEIN thought it was apparent that the tonsillectomy had nothing to do with the hyperpyrexia, for the condition is known to occur following various surgical operations anywhere in the body, particularly in cleft palate cases in which postmortem examination has shown extreme hyperemia of the brain. He recalled a case which followed an hypophyseal operation under local anesthesia. This was one of a series of eighteen cases, and resulted in death from hyperpyrexia in two days. The operation was very rapid and uncovered an enormous cyst. Through some mistake the incision was enlarged very rapidly, and a large amount of straw colored fluid escaped. No attempt was made to enter the cyst, but the man within a few hours developed a high temperature, which reached 107.5° F. before death occurred, two days later. When seen on the evening of the operation a strong odor of acetone could be detected on his breath. Dr. Stein thought that possibly the fatal termination was due to the sudden release of pressure at the base of the brain, in the neighborhood of the heat regulating mechanism.

DR. JOHN A. CAVANAUGH cited a case seen with Dr. Tydings in a young lady who developed hemorrhage following operation. She was taken to the Chicago Eye, Ear, Nose and Throat Hospital, and within six hours developed a temperature of 105° F., which persisted for two days and was accompanied by delirium. After the second day the temperature dropped to almost normal and remained there for twenty-four hours, when it rose to 104.5° F., where it stayed for about thirty-six hours and then subsided. Nothing was ever found to explain the cause. The patient recovered.

DR. J. GORDON WILSON said he had seen several cases of hyperpyrexia, but none associated with removal of the tonsils. Hyperpyrexia he divided into two groups—one coming on immediately after an operation and the other developing after



some days. The first he considered due to disturbance of the heat regulating mechanism, the delicate nervous mechanism which keeps warm blooded animals at an even temperature. In such cases the operation, for an unknown cause, throws the mechanism out of action—comparable in some way to "shock"—and the temperature rises abnormally. In some on post-mortem examination hyperemia of the brain has been disclosed, but in others no postmortem change could be demonstrated.

In the second group there is an abrupt rise of temperature later; it may be on the second or third day after the operation. This, he believed, is due to some toxic absorption.

He has seen in children, following a tonsil operation, a rise of temperature which may be abnormally high, but in no case has he seen anything approximating a hyperpyrexia or a fatal result.

DR. HOWARD C. BALLENGER said that in order to determine the question of bacteremia following tonsillectomy at the Sprague Institute of the Children's Memorial Hospital, a series of seventy-three blood cultures were taken by Dr. Rubin during and after the tonsil operation to see whether fever following tonsillectomy could be explained through a bacteremia. In all seventy-three cases the cultures proved negative. He thought it apparent that a bacteremia does not have much to do with these cases, but due allowance should be made because of the difficulty of determining whether a bacteremia is present, especially a transitory bacteremia.

DR. NOAH SCHOOLMAN thought that the adenoidectomy is probably as frequently implicated in these rare accidents as the operation on the tonsils. The vault of the pharynx is attached to the most vulnerable part of the base of the skull, involved in this operation. On its cerebral aspect lie the medulla and the pons with the cranial nerves emerging from them. It is also in close proximity to the hypophysis. In intra-uterine life this portion of the basic cranii, the pars basilaris, exists as a separate mass of embryonic osseous structure with loose cartilaginous attachment to the rest of the base of the skull. These conditions may persist indefinitely into infancy and childhood. Adenoidectomy, as usually performed, with

massive instruments which exert considerable force upon this portion of the cranium which may, under unusual predisposing circumstances, cause hemorrhage or other injury to the superimposed midbrain.

He cited the interesting work of Professor Levy of Berlin regarding the vascular and lymphatic relations of the adenoid and the hypophysis and the pathologic implications of such relations under some circumstances.

DR. ARTHUR M. CORWIN asked if there was any clinical record of the treatment of hyperpyrexia in these cases.

DR. JOSEPH C. BECK, referring to acidosis following the administration of nitrous oxid anesthesia, cited a case in which this anesthetic was given to a patient on whom they started to operate under local anesthesia. The patient was a woman, aged 60, who had difficulty in breathing because of a central thyroid gland. They did a local infiltration with apothesine, the object being to do a tracheotomy to give room for breathing. After completing the infiltration and dissecting freely the gland they had great difficulty in keeping the patient quiet, so nitrous oxid gas was given in order to expose the trachea. The patient breathed easily immediately, but promptly went into a condition of acidosis. Her breath was very strong, she vomited, developed all the symptoms of acidosis and died within a few hours. There were no symptoms referable to the lungs and a postmortem could not be secured.

In the investigation of the condition of hyperpyrexia he had hoped to hear something of the work of Vaughan, who did a great deal in the way of producing high temperatures by giving foreign proteins.

DR. SAMUEL J. PEARLMAN, in closing, said the object of the paper was to call attention to a syndrome of high temperature, pallor and occasional death. Because of the few cases reported they thought it was an extremely rare condition, but judging by the cases cited in the discussion it was evidently more common than they had believed.

The fact that thrombi are found in the tonsillar fossa following tonsillectomy had nothing to do with it, but minor operations under general anesthesia, and sometimes under local, do produce this syndrome. There is no agreement as to the

cause, but he thought the suggestion of Dr. Wilson, that there was some connection with the heat regulating mechanism, was to the point. The pallor is that associated with bulbar involvement. Those cases of sepsis following tonsillectomy usually survive for a number of days, and postmortem findings often indicate the cause of death, and show, for instance, thrombi in the jugular. As to split proteins causing it, he thought no one could say.

The lesson to be learned from these cases was that no operation should be undertaken unless there was a very good reason for it.

#### Foreign Body in the Lung.

By JOHN A. CAVANAUGH, M. D.

This case was presented because of the unusual location of a foreign body in the lung.

Mrs. E. S., aged 52, was referred because of a probable foreign body in the right lung, with the following history:

While eating chicken at dinner, the night before, she suddenly choked, gasped for breath and ran to the back porch seeking air; she stuck her finger into her throat, something gave way and she gradually regained breath and felt better. There was very little coughing but a slight pain developed in the right chest. Convinced now that a foreign body was there, she assumed various positions, almost standing on her head forward, hoping gravity might assist in getting rid of it. Finally a neighborhood doctor was consulted, who assured her that anxiety was needless, but not being satisfied she sought the advice of a throat specialist, who examined her throat and also declared there was nothing wrong. She returned home, still feeling the discomfort, went to bed, passed a restless night; feeling a tightness in the chest, began to wheeze, and feared she had asthma. The following morning another doctor was consulted, who thought a foreign body might be in the lung. A radiogram was made which showed a rather long, thin shadow in the right chest, and she was referred to Dr. Cavanaugh, who saw her about 2 p. m. There were distinct râles over the right lung. Temperature 99° F., pulse 100, respiration 24. The right vocal cord had on its upper surface a submucous hemorrhagic spot in its middle third. The balance of

the larynx was a dusky red and the arytenoids were slightly edematous. He advised going to the hospital at once, but met with considerable opposition from the patient and the daughter. Mrs. E. S. cited a case reported in the newspaper where a patient coughed up a screw after being held up by the feet, and wanted to know if he couldn't hang her up by the feet and shake it out. After much controversy she went to St. Luke's Hospital. A quarter grain of morphin sulphate and 1/200 gr. of scopolamin hydrobromid were given hypodermatically, applications of 10 per cent cocain to the larynx and an application of 5 per cent below the glottis. He introduced a 7 mm. Brunning tube and when the right main bronchus was reached he could see a somewhat whitish mass on the anterior wall. Passing into the right bronchus he could see the foreign body lodged in the mouth of the middle lobe and with a Jackson forcep he grasped the protruding portion and removed it. The size necessitated the removal of the tube at the same time. When the glottis was reached he turned the foreign body so the long axis would be with the long axis of the glottis and it slipped through without traumatizing. The object was a chicken bone, 2 mm. long, 13½ mm. thick at one end and 10 mm. at the other. The patient was removed to a croup tent, where she remained three days, making an uneventful recovery. When he saw her at the office a week later, a little redness of the cords remained; otherwise they were normal and the chest showed negative findings.

DR. EDWIN MCGINNIS referred to a case in which a child coughed up a screw, and said Tucker had told him that only about 2 per cent of the foreign objects in the finer bronchi were coughed up. He congratulated Dr. Cavanaugh on getting such a large object out between the cords without injury to them. Recently a dentist, while attempting to fit a five-tooth bridge to some prepared teeth, let it slip and it landed in the right bronchus. It took a good deal of patience to slide it out between the vocal cords without injury to them.

DR. GEORGE W. BOOT stated that in the last three cases he had treated bronchoscopically the patient had coughed up the foreign body before he could reach it. He considered it a very serious matter to take out a large foreign body between the cords, particularly if it was larger than the tube. In a case seen

recently in which a child had swallowed half a peanut kernel, the patient died before he could perform a tracheotomy.

**Foreign Body (Deposit of Barium) in Antrum.**

BY AUSTIN A. HAYDEN, M. D.

The patient was a school teacher who had been operated upon for the removal of gallstones. Her nose and throat were examined before operation and found to be negative. A few days after the cholelithotomy she complained of pain in the region of the right antrum. A skiagram revealed the presence of a foreign body. Investigation showed this to be a mass of barium that had lodged in the antrum when the patient vomited following a barium meal during the examination of the gastrointestinal tract.









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